THE American JOURNAL OF Surgery

AUGUST 1957

THURSTON SCOTT WELTON, M.D., EDITOR

Papers of the
Pacific Coast Surgical Association

SIXTY-SIXTH YEAR OF PUBLICATION

AUGUST 1957

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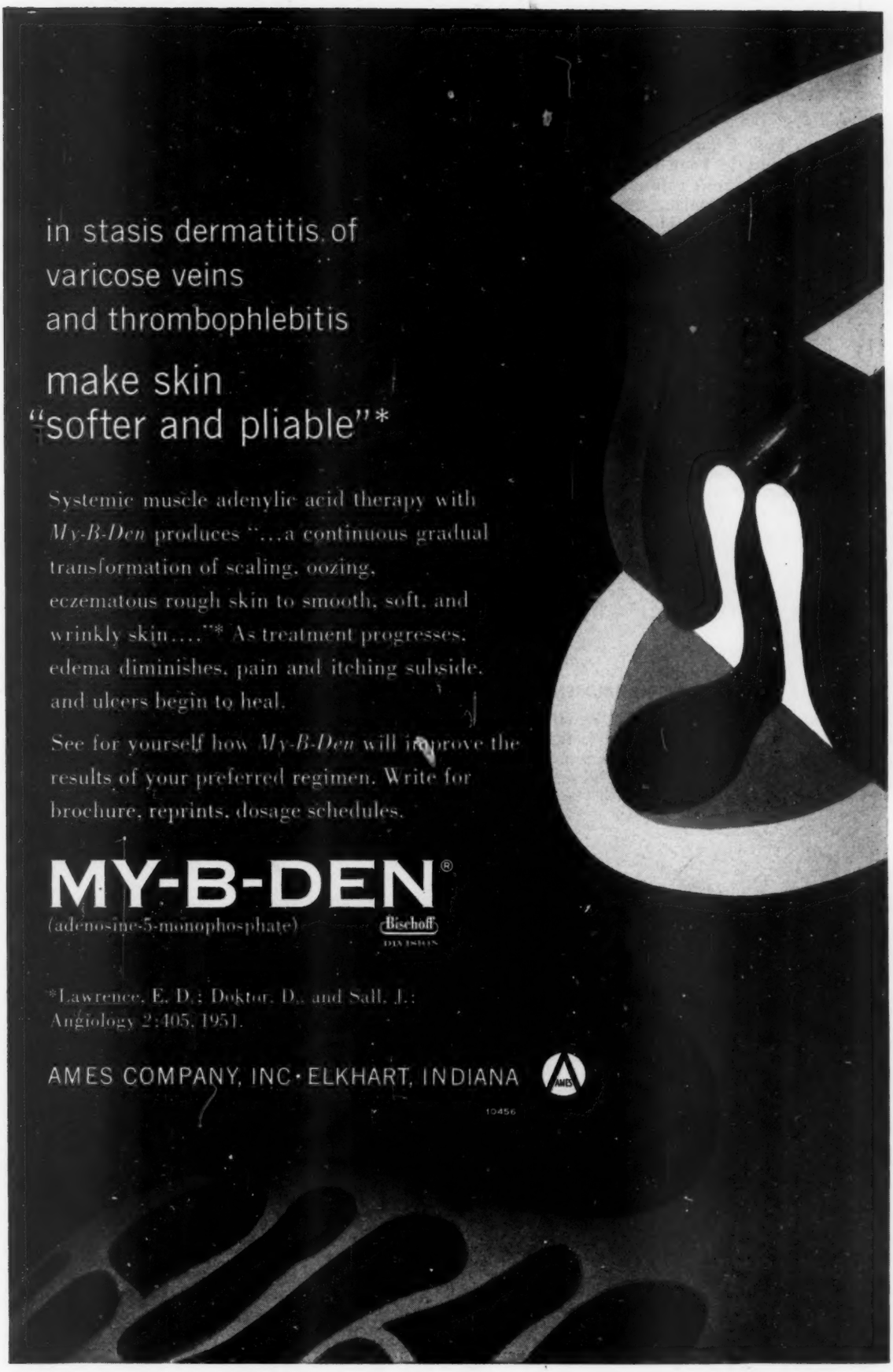
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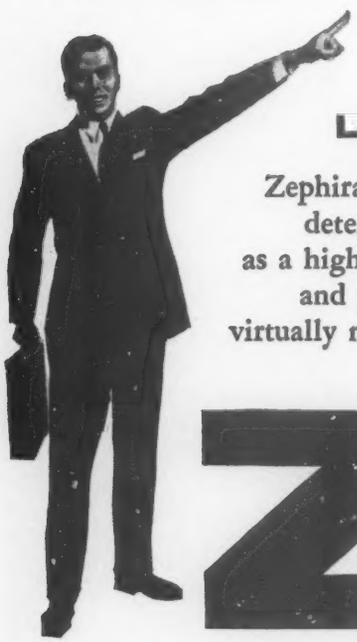

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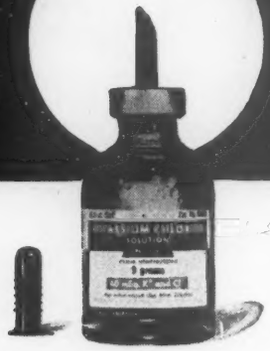

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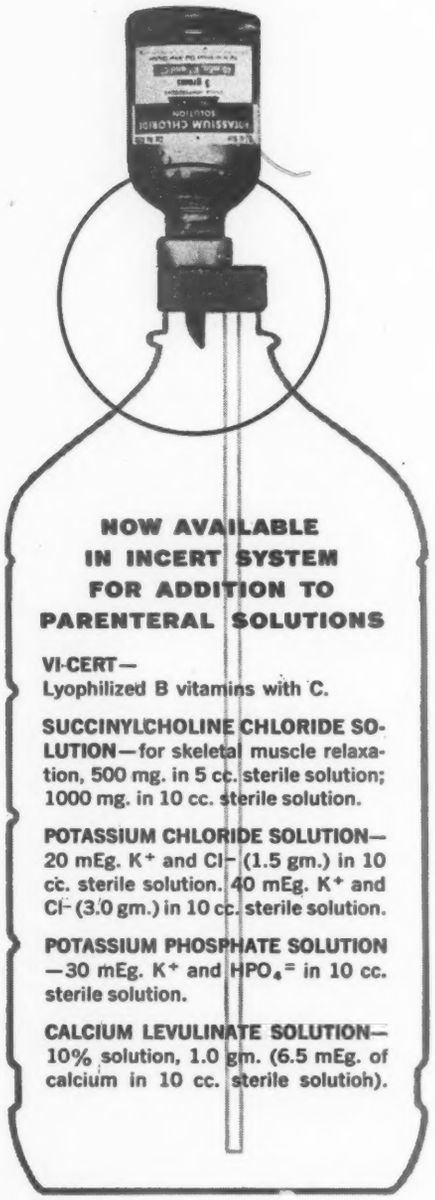

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[1] Miller, J. M.; Surmonte, J. A.; Ginsberg, M., and Ablondi, F. B.: Streptokinase Intramuscularly in the Treatment of Infection and Edema. (Scientific Exhibit) *Postgraduate Medicine* Vol. 20, No. 3: 260-267 (Sept.) 1956.

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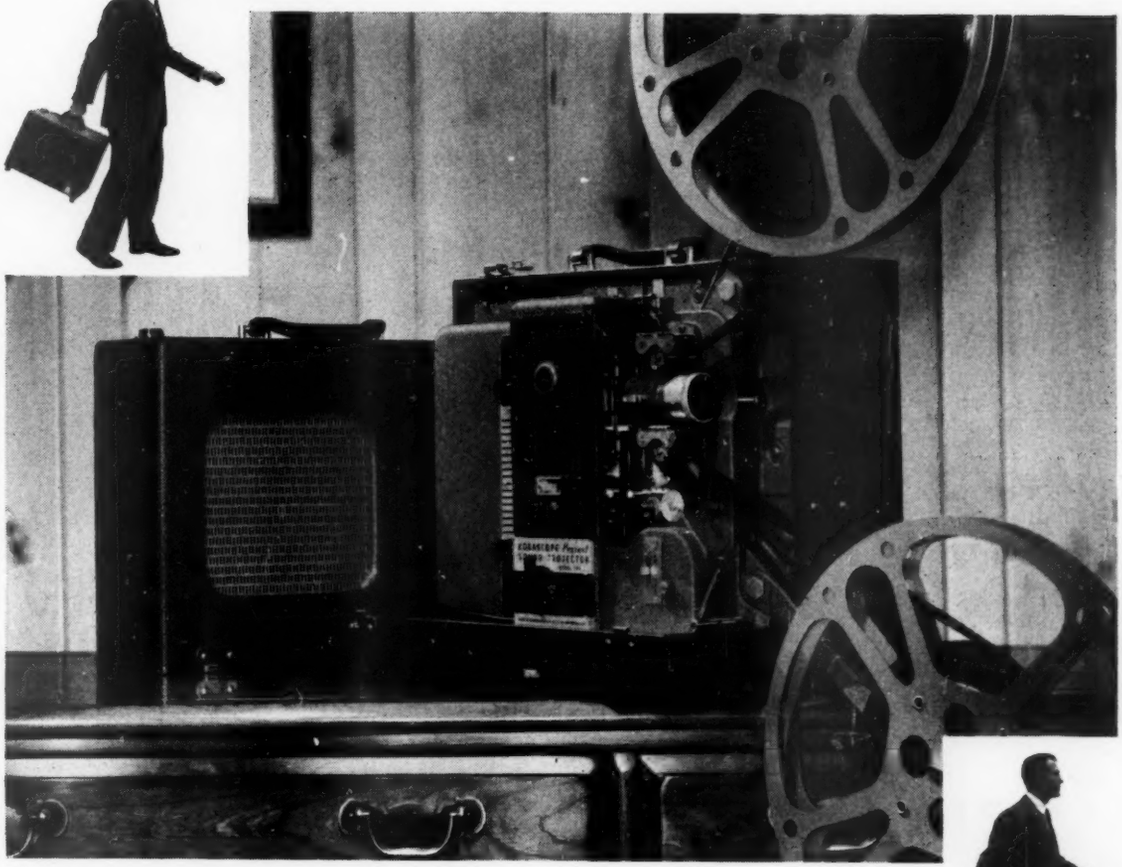

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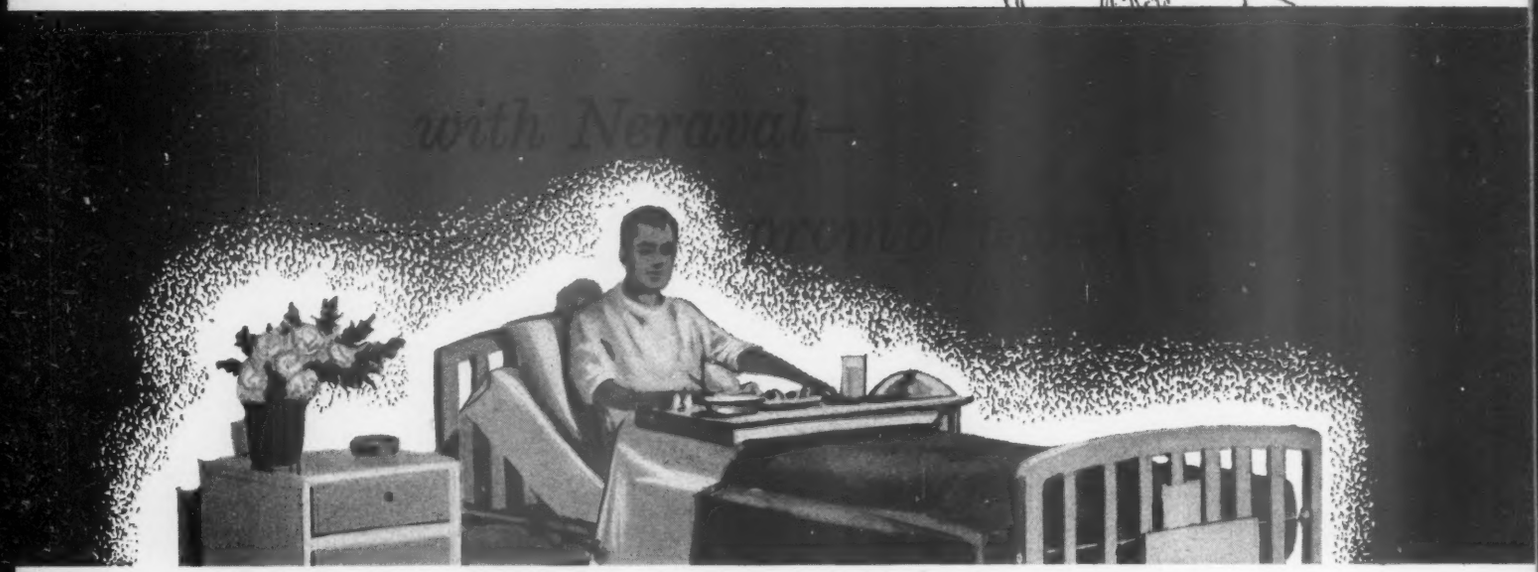

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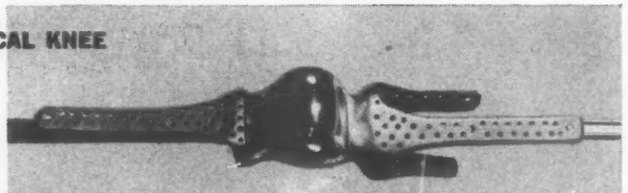

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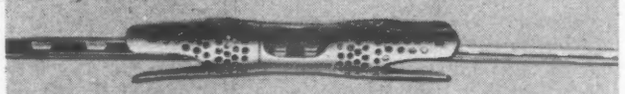

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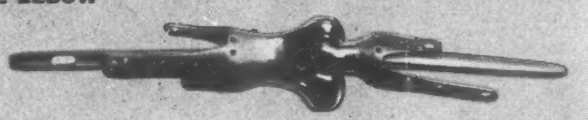

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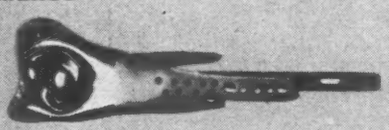

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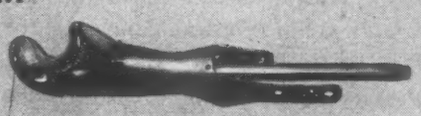

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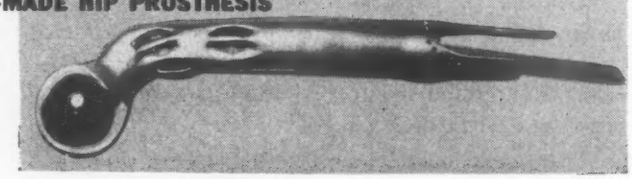
One of many designed by surgeons to meet individual requirements where standard designs do not fit.



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AUSTENAL, INC. : SURGICAL DIVISION

224 EAST 39th STREET • NEW YORK 16, N.Y.

for severe infections
new, lifesaving

FURADANTIN®

BRAND OF NITROFURANTOIN

Intravenous Solution

often rapidly effective

- **in bacteremia, peritonitis, and other bacterial infections as of postoperative wounds and abscesses, when the organism is susceptible to Furadantin**
- **in severe genitourinary tract infections when the patient is unable to take Furadantin per os**

Representative cases of severe urinary tract and extra-urinary tract infections in which intravenous administration of Furadantin achieved clinical and bacteriologic cure or marked improvement in a high percentage.

Case Summaries

Sex; Age; Disease Treated	Causative Bacterium	Coexisting Disorder
M; chronic cystitis, pyelonephritis	Ps. aeruginosa in urine	---
F; chronic pyuria with fever	E. coli in urine	---
F; 39; pyelonephritis	Ps. aeruginosa in urine	Staghorn calculus, right
M; 79; bacteremia	E. coli in urine	Post-trans
F; pyuria, probable bacteremia		
M; pyelitis, probable bacteremia, chills		
Bacteremia		
M; empyema and post-surgical wound infection		
M; 75; acute pyelonephritis	pus in urine	month before
F; 77; cystitis, atypical pneumonia	(Not stated)	Diabetes mellitus
F; furunculosis	M. pyogenes var. aureus	Post-pyelonephritis

(52%) (39%) (8%)

(50%) (33%) (16%)

(52%) (26%) (22%)

(22%) (61%) (16%)

(25%) (53%) (21%)

(34%) (45%) (20%)

FURADANTIN I.V.

Furadantin I.V. Dosage	Result of Treatment	Remarks
180 mg. daily, 3 days	Cured	Resistant to broad spectrum antibiotics and sulfonamides. In 3 days, afebrile; urinary frequency, urgency gone, urine culture negative.
180 mg. daily, 4 days	Cured	Resistant to penicillin and sulfonamides. In 4 days, "afebrile and infection cleared," urine culture negative.
60 mg. q.i.d.	Cured	Organism resistant in vitro to Furadantin. Changed to oral form after 3 days on I.V.
	Cured	36 hours post-op. temp. 106.4°. Remission of fever 16 hours after starting Furadantin I.V. Negative urine culture in 5 days.
	Cured	Antibiotics for 2 weeks. Added oral Furadantin 4 days. Patient semicomatose when Furadantin I.V. started. Afebrile in 24 hours. Urine culture sterile at 14th day.
	Cured	When Furadantin I.V. started temp. 104°. In 24 hours, temp. 99°. Previous treatment included penicillin, streptomycin and sulfonamide for 5 days each. Patient discharged on oral Furadantin for 5 days. Returned to clinic with negative urine.
	Inconclusive, blood culture negative	"Patient had been resistant to penicillin and streptomycin." Chlortetracycline given intercurrently with Furadantin I.V.
	Improved	Previous streptomycin and INAH, pneumonectomy. Empyema and wound infection with Pseudomonas. After Furadantin I.V. temp. normal, wound improved. Continued on oral Furadantin.
180 mg. b.i.d., 2 days	Cured	Temp. 103° reduced to normal, urine clear and patient discharged in 2 days. ". . . without Furadantin I.V. . . . probably would have died."
180 mg. daily, 3 days	Cured	Penicillin-dihydrostreptomycin 5 days previous. Temp. 104.4° to normal 24 hours after Furadantin I.V. instituted.
180 mg. daily, 3 days	Markedly improved	Continued elevation in temp. (100-101°) for 10 weeks in spite of intensive antibiotic therapy. Temp. dropped slowly to normal on Furadantin I.V. and no new furuncles developed.

FURADANTIN *Intravenous Solution*

DOSAGE AND ADMINISTRATION: Furadantin Intravenous Solution contains 0.6% Furadantin, dissolved in polyethylene glycol 300. Each 10 cc. ampule contains 60 mg. of Furadantin. The solution is sterile and must be dissolved aseptically in a sterile diluent prior to use.

The recommended diluent is 5% dextrose solution. Furadantin Intravenous Solution is compatible with normal saline and ⅙ M sodium lactate solutions, but these should be used only when definitely indicated because of the possibility of further disturbing an already upset electrolytic balance. Administer as an intravenous drip at a rate indicated for the patient's general condition and age. The suggested dosage is 5 to 7 mg. per Kg. body weight (2.2 to 3.2 mg./lb.) per 24 hours in 2 divided doses. The single dose of Furadantin Intravenous Solution for the average adult is 30 cc. (3 ampules or 180 mg.) in at least 500 cc. of diluent. If tolerated, this is repeated so that 2 such doses (360 mg.) are given over 24 hours and continued for 7 days, if necessary. Following are average dosage schedules:

Weight of Patient	*Size of Single Dose*	*Minimal Amount of Diluent*
120 lbs. or over (54 Kg.)	30 cc. (3 amp.) = 180 mg.	500 cc.
80 to 120 lbs. (36-54 Kg.)	20 cc. (2 amp.) = 120 mg.	350 cc.
40 to 80 lbs. (18-36 Kg.)	10 cc. (1 amp.) = 60 mg.	175 cc.

In urinary tract infections, the intravenous form may be replaced by the oral dosage form of Furadantin when sufficient improvement has resulted and patient can take medication orally.

SIDE REACTIONS: Nausea or emesis may occur occasionally. These are often minimized by a decrease in dose rate. Sensitization occurs rarely in the form of urticaria or an erythematous, maculopapular rash. This may be controlled by discontinuing treatment immediately and employing the usual measures such as epinephrine, antihistaminics or adrenocorticosteroids. Occasionally patients may show minor side reactions such as headache or malaise. No stomatitis, colitis, proctitis, anal pruritus, monilial superinfection, staphylococcic enteritis or renal, hepatic or hemic toxicity have been reported.

With intravenous administration, it is important to be alert to the possibility of central nervous system effects such as muscular twitching or spasticity. Should this occur, calcium gluconate should be administered intravenously and Furadantin discontinued. Adequate amounts of vitamin B complex and vitamin C should be supplied to the patient on intravenous Furadantin therapy.

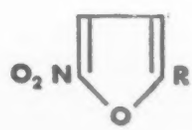
SUPPLIED: Sterile 10 cc. ampules (60 mg. Furadantin each), box of 12.

NOTE: Furadantin Sensi-Discs (100 mcg.) for sensitivity testing are available from the Baltimore Biological Laboratories. Materials and instructions for serial-tube dilution tests may be obtained from the Medical Director of Eaton Laboratories. To simplify sensitivity testing with Furadantin and to provide maximum reproducibility and reliability, Eaton Laboratories has prepared a new booklet, "Bacterial Sensitivity Testing with Furadantin and Furacin," copies of which may also be obtained by writing to the Medical Director.

For oral administration in the treatment of genitourinary tract infections: Furadantin tablets, 50 and 100 mg.; Furadantin Oral Suspension, 25 mg. per 5 cc. tsp.

NITROFURANS–*a* new *class of antimicrobials—neither antibiotics nor sulfonamides*

PRODUCTS OF EATON RESEARCH—EATON LABORATORIES, NORWICH, N. Y.

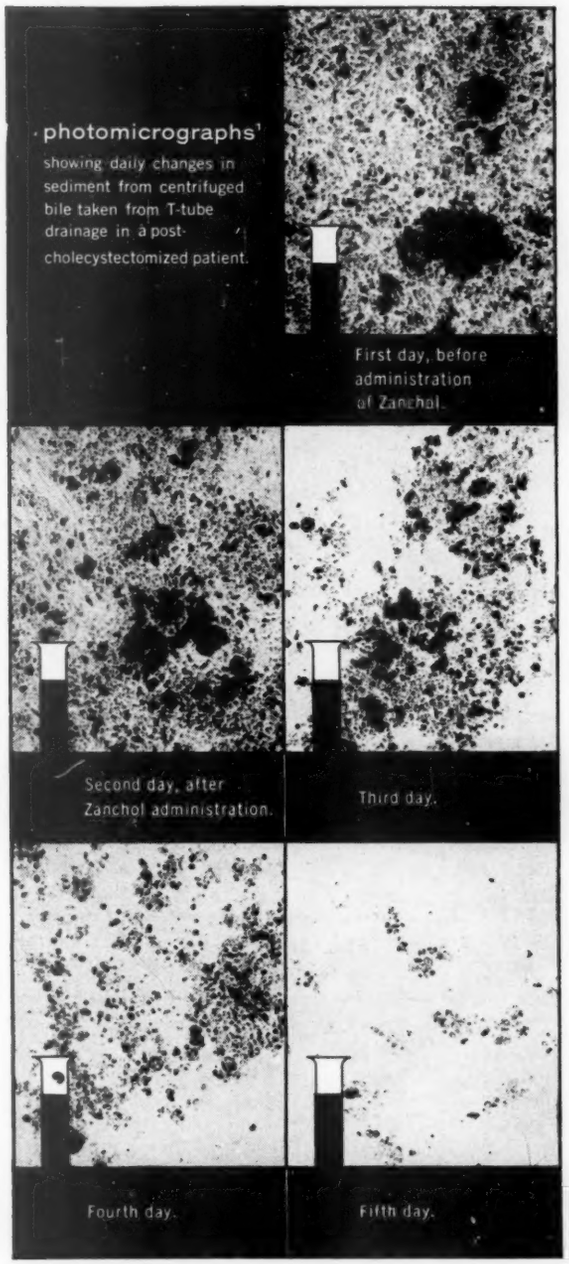


NEW SEARLE RESEARCH PRODUCT

ZANCHOL*

(brand of florantyrone)

Synthetic Biliary Abstergent Fills an Important Postcholecystectomy Need


photomicrographs[1]
showing daily changes in sediment from centrifuged bile taken from T-tube drainage in a post-cholecystectomized patient.
First day, before administration of Zanchol.
Second day, after Zanchol administration.
Third day.
Fourth day.
Fifth day.

The excellent results with Zanchol in patients whose gallbladders have been removed have been most pronounced in two phases of management:

1. Early—Zanchol in Postoperative Care. T-tube studies have demonstrated that Zanchol increases the volume and fluidity of bile, at the same time changing its color to a clear, brilliant green. The greatly improved abstergent cleansing action of the bile is noted in its ability to keep the T tubes clean[1] without rinsing in most cases.

2. Late—Zanchol in Postcholecystectomy Syndrome. By improving the physicochemical properties of bile and increasing its flow, Zanchol acts to eliminate biliary stasis and sharply reduce or eliminate biliary sediment. The drug may be employed in both prophylaxis and therapy of the postcholecystectomy syndrome.

Medical Indication for Zanchol

This includes the treatment of patients with chronic cholecystitis for which surgery is not required or may be impossible for any reason.

Dosage: one tablet three or four times daily.

Supplied: Zanchol is available in tablets of 250 mg. each.

G. D. Searle & Co., Chicago 80, Illinois.
Research in the Service of Medicine.

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1. McGowan, J. M.: Clinical Significance of Changes in Common Duct Bile Resulting from a New Synthetic Choleretic, Surg., Gynec. & Obst. *103*:163 (Aug.) 1956.

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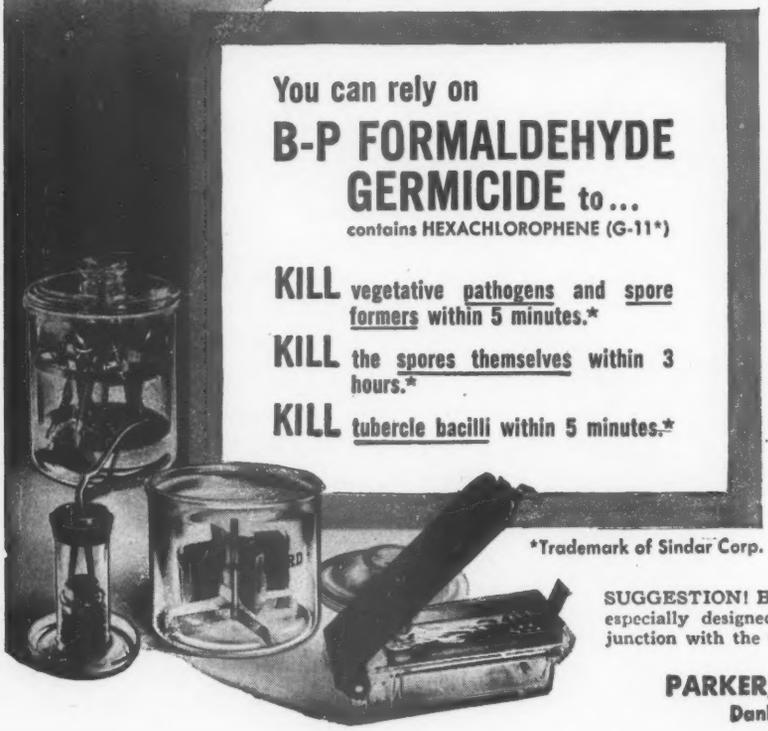
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NEW INTRAMUSCULAR IRON PROVIDES PRECISION THERAPY, PROMPT RESPONSE

IMFERON,® the new intramuscular iron-dextran complex, was introduced to American hematologists at the Sixth International Congress of the International Society of Hematology held in Boston, August 27 to September 1, 1956. Recent experience from over 6 million injections has shown that this iron preparation is easy to administer, notably free from toxic effects, quickly absorbed and productive of rapid hematologic and clinical improvement. It has been termed "...the only therapeutically effective iron preparation for intramuscular use...."[1]

IMFERON meets the need for a safe, effective agent when parenteral iron is preferable for patients with iron deficiency anemia who are resistant or intolerant to oral iron, those with depleted iron reserves and those who require rapid restoration of hemoglobin, *e.g.* last trimester of pregnancy.

Previous parenteral iron preparations were unsatisfactory because of toxicity, pain on injection, or because they contained insufficient iron. IMFERON contains the equivalent of 5 per cent elemental iron. It is more stable than iron saccharate both *in vitro* and *in vivo* and does not precipitate in plasma over a wide pH range. It is isotonic with tissue fluids and has a pH of 5.2 to 6.0[1] Utilization for hemoglobin formation is almost quantitative.

Precision Therapy with IMFERON: Before treating a patient with IMFERON, total iron requirement is calculated by formula or determined from a convenient dosage chart. Then appropriate amounts of IMFERON are injected daily or every other day, until the total calculated required amount is given.

Iron Deficiency Anemia of Infancy: IMFERON provides a convenient safe means for restoring hemoglobin levels and iron reserves in anemic infants. Excellent results were obtained by Gaisford and Jennison[2] with IMFERON in 100 iron-deficient infants. From a pretreatment average of 54.5 per cent, hemoglobin levels rose to 87 per cent 10 weeks after the start of therapy. Clinical improvement paralleled this response. Premature infants and surgical cases were similarly benefited. IMFERON gave "...all the advantages of transfusion or intravenous therapy without the disadvantages."[2] There were no side effects in any of the infants treated. Wallerstein[3] confirmed these results, furnishing evidence that IMFERON is well absorbed and appears in the bone marrow 12 to 24 hours after injection. Results are equal to those with intravenous saccharated iron oxide without the unpleasant side effects. Sturgeon[4] showed that the first year's iron requirements in infancy can be supplied with three injections of IMFERON.

Iron Deficiency Anemia of Pregnancy: Nausea precludes oral iron therapy in many anemic pregnant women. In those with severe anemia who are first seen late in pregnancy, prompt hemoglobin regeneration is unobtainable with oral iron. IMFERON produced prompt hemoglobin responses in anemia of pregnancy,[5,6] the results being similar to those obtained with intravenous saccharated iron oxide. Side effects were virtually absent with IMFERON.[5,6]

Resistant Hypochromic Anemia: Patients who do not respond to oral iron, those who cannot take oral iron and those with gastrointestinal pathology respond well to injections of IMFERON.[7-11] While oral iron is of little value in treating the anemia of rheumatoid arthritis, IMFERON is "...as beneficial as intravenous iron and easier to administer."[9]

Present Studies: Published reports and recent findings of clinical investigators confirm the effectiveness and safety of IMFERON for hemoglobin regeneration and creation of iron stores. More than 70 studies are now being completed in the United States. Reports stress prompt hemoglobin response, ease of administration and freedom from side effects. Clinicians desiring additional information should request Brochure No. NDA 17, IMFERON, Lakeside Laboratories, Inc., Milwaukee 1, Wisconsin.

References: (1) Brown, E. B., and Moore, C. V., in Tocantins, L. M.: Progress in Hematology, New York, Grune & Stratton, Inc., 1956, vol. I, p. 25. (2) Gaisford, W., and Jennison, R. F.: Brit. M. J. *2*:700 (Sept. 17) 1955. (3) Wallerstein, R. O.: J. Pediat. *49*:173, 1956. (4) Sturgeon, P.: Pediatrics *18*:267, 1956. (5) Jennison, R. F., and Ellis, H. R.: Lancet *2*:1245 (Dec. 18) 1954. (6) Scott, J. M., and Govan, A. D. T.: Brit. M. J. *2*:1257 (Nov. 27) 1954. (7) Grunberg, A., and Blair, J. L.: A.M.A. Arch. Int. Med. *96*:731, 1955. (8) Millard, J. B., and Barber, H. S.: Ann. Rheumat. Dis. *15*:51, 1956. (9) Baird, I. M., and Podmore, D. A.: Lancet *2*:942 (Nov. 6) 1954. (10) Cappell, D. F.; Hutchinson, H. E.; Hendry, E. B., and Conway, H.: Brit. M. J. *2*:1255 (Nov. 27) 1954. (11) Stevens, A. R.: A.M.A. Arch. Int. Med. *96*:550 1956.

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On the Utilization

of Oatmeal Carbohydrate

One of the distinguishing characteristics of oats is its relatively lower content of carbohydrate and its higher content of protein, when compared with other whole-grain cereals.[1] An important feature of the carbohydrate of oatmeal is its mode of utilization by the body.

Because it consists mainly of slowly available polysaccharides with relatively minor amounts of dextrins and simple sugars, the carbohydrate of oatmeal does not tend to cause a sudden, sharp increase in blood sugar. Instead, the complex carbohydrate molecules gradually release glucose into the blood stream and thus provide energy on a more sustained basis. Oatmeal is therefore logically recommended as an excellent breakfast dish for diabetics.[2]

The carbohydrate content of oats is almost completely digested and utilized. Cellulose, or cellulose-like material, representing only about 2 per cent of rolled oats in the dry form, provides a gelatinous residue which serves advantageously as nonirritant bulk in the lower intestine.

Whether eaten for therapeutic reasons during illness or for a good sturdy breakfast every day, oatmeal and milk (skim or whole) assures high biologic quality protein, vitamins, and essential minerals, in addition to ease of nutritional utilization.

Quaker Oats and Mother's Oats, the two brands of oatmeal offered by The Quaker Oats Company, are identical. Both brands are available in the Quick (cooks in one minute) and the Old-Fashioned varieties which are of equal nutrient value.

1. Kent-Jones, D.W.: Modern Cereal Chemistry, ed. 3, The Northern Publishing Co., Liverpool (1939).
2. Heupke, W., and Kolb, A.: Mechanism of the Beneficial Action of Alimentation of Oats in Diabetes, Deutsche Ztschr. Verdauungeskr. *13*:111 (1953).

IN POSTSURGICAL NUTRITION oatmeal finds a well-merited place as soon as feedings no longer need be limited to clear liquids. Its high nutrient value and blandness aid in rapid, uneventful re-alimentation.

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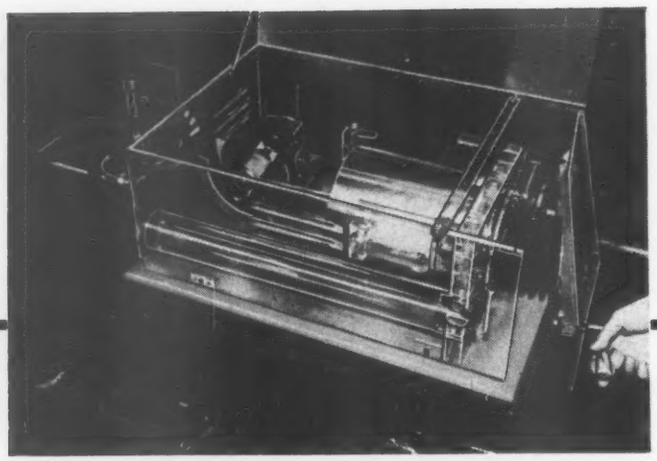

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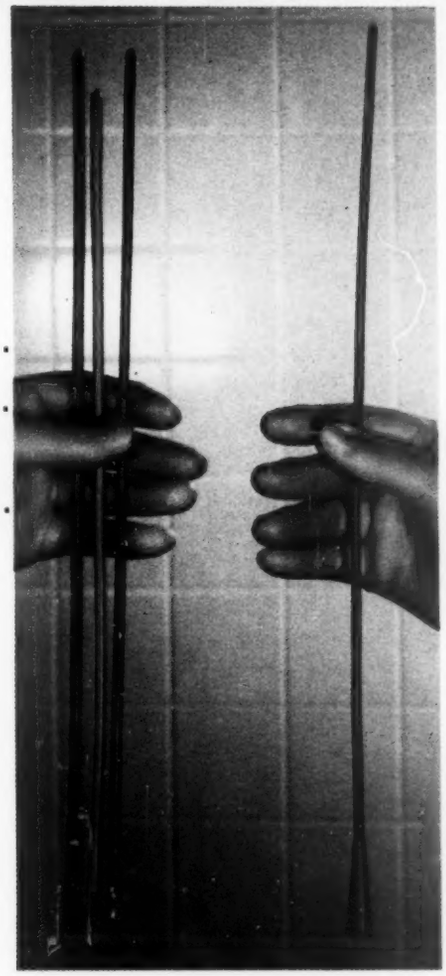

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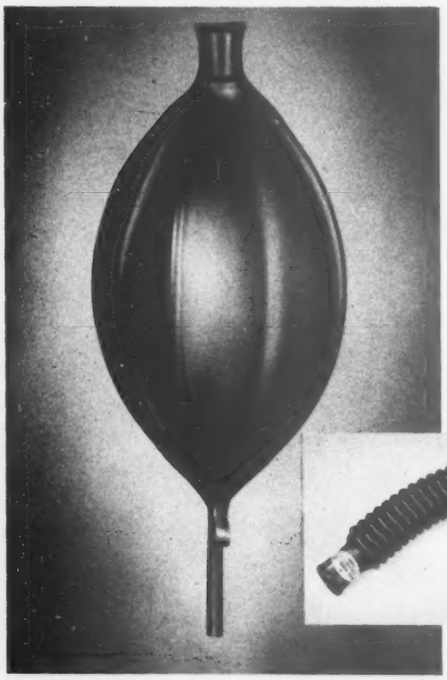

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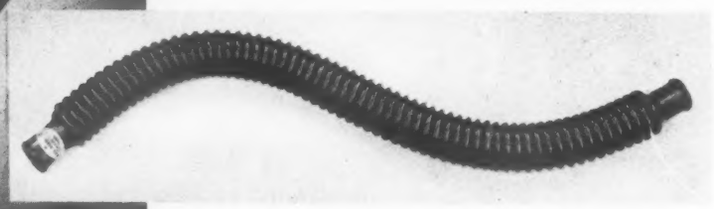

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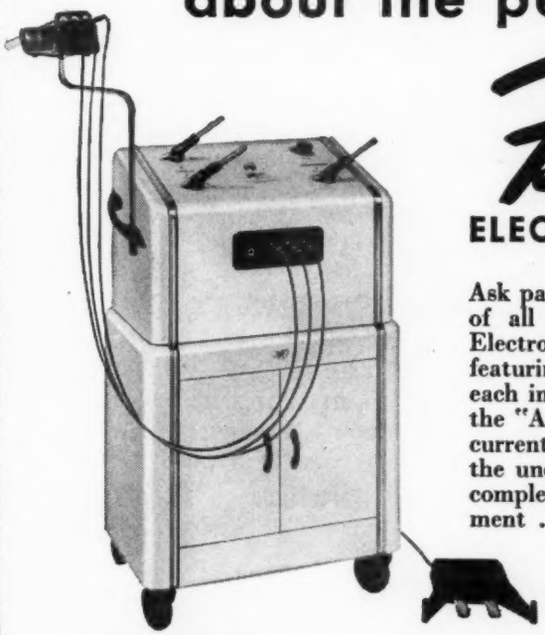

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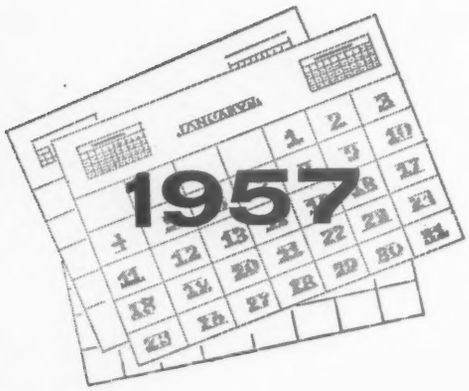

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SUMYCIN
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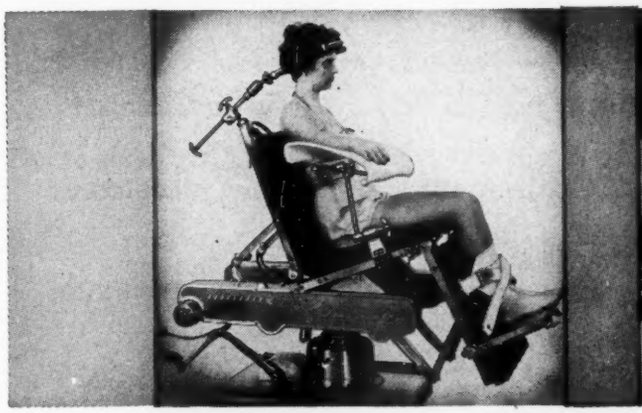

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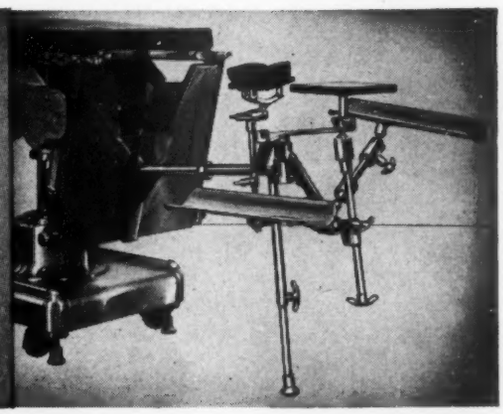

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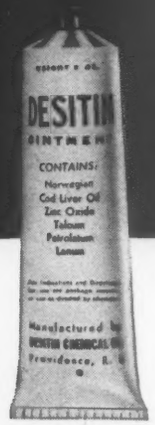

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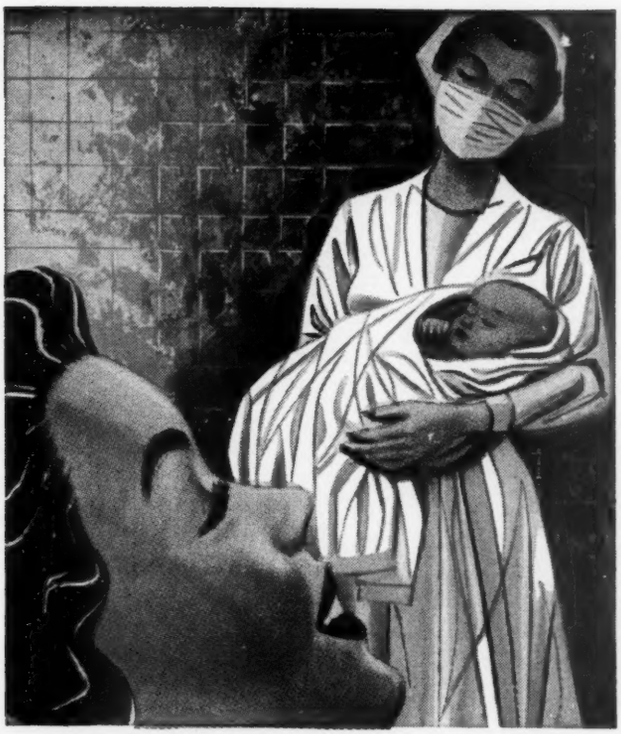
1. Grayzel, H. G., and Schapiro, S.: Western J. Surg., Obstet. & Gynec., Oct. 1956.



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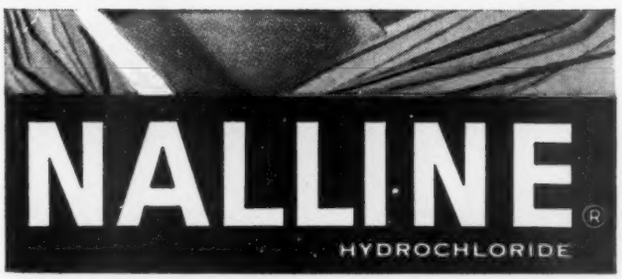

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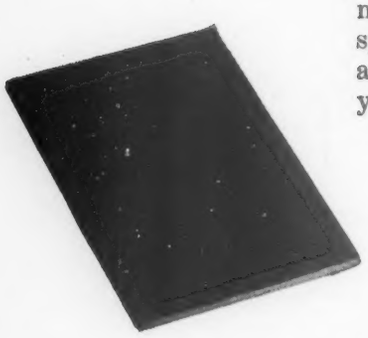

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The American Journal of Surgery

VOLUME 94 AUGUST 1957 NUMBER TWO

PRESIDENTIAL ADDRESS

A Response to Questions from Younger Surgeons

I STAND before you a humble man. Many could more adequately represent this Association than I, but let me assure you that this high honor could not be more greatly and sincerely appreciated. In this grateful and, I must confess, somewhat timorous mood, I have decided to speak as I would to a younger man in this Association who might ask me what his aims should be as he approached the most productive years of his life. These aims, I would tell him, are bound up with a series of obligations, difficult and demanding, which we may ignore only at our peril.

As surgeons, we are singularly lucky men, well placed in a free society and privileged with an unparalleled opportunity to serve. Ours is the chance to exemplify free private enterprise at its best, disciplined by science and humanized by the fundamentally compassionate nature of our work. Because we are so fortunately situated, I think most of us feel that we owe something. What? To whom? Each of us will have his own ideas; here are a few of mine.

First of all, we owe ourselves good health. Through the centuries the world has snickered at bald barbers and sick doctors. The physician becomes too busy taking care of his patient to take care of himself. Our occupation is disease, and our occupational disease is overwork. Insofar as our profession demands the sacrifice of our own health, we must make that sacrifice. But do any of us make needless sacrifices? We, of all people, should properly respect our bodies, but do we always do so? Or does the detachment with which we must examine our patients sometimes so infect us that we ignore our own symptoms? Our health is not a matter of purely selfish significance. If we do not listen to our own doctors, we penalize our society with an incalculable and irrevocable waste—medical, economic and social. Our country cannot afford to lose a good surgeon in his prime because he has neglected to look after himself.

I have no easy solution. "Do not work so hard" will not do. The better the surgeon, the greater the demands upon his time. As the years advance upon us, however, we can perhaps lighten our burdens by sharing them. There must come a time when we relinquish some of our activities. Surely the best time is not when we are on the downgrade, not when we have to be helped, but while we can still give help. Many young surgeons with adequate qualifications would be glad to work with an established man. Would it not be wise to recognize the mutual advantages of such an association?

So much for ourselves. What do we owe our families? The cold soup, the warmed-up chop, the empty chair—at the fireside and in the theater—symbolize our profession as accurately as the caduceus. For her understanding and fortitude, her constant patience and conspicuous gallantry above and beyond the call of duty, the surgeon's wife deserves a daily medal; some days she is lucky if she gets a hurried "hello." How can we make up for the

empty evenings, the emergencies and interruptions to which we have subjected her? With some trepidation, may I make one or two suggestions?

Life magazine, a few months ago, described the attempts of the Air Force to arouse the interest of pilots' wives in their husbands' duties by putting the wives through a modified routine of flight training. I do not mean to suggest that we should ask our wives to perform an occasional appendectomy or assist us in a particularly interesting gastric resection. But I do suggest that we can, when asked about our day, return a more informative answer than "fine" or "so-so." We should let our wives share a little of the world that keeps us so much apart. We can do more. We can try even harder to understand our wives. The demands we must make we can mollify with courtesy, freely and wholeheartedly given. We can be just a little more considerate than other men, in private and public. When alert enough to try, I have been well rewarded.

The surgeon has the further responsibility of providing for his family. Let me cite a short, sad story. Dr. X, a respected, successful surgeon, died unexpectedly at the height of his career, leaving nothing, not even an insurance policy. The house, the beach cottage, an automobile, a motorboat and expensive furnishings all had to be sold. The wife now has her first job; the three younger children are working their way through college, helped by gifts and grants-in-aid. Surgeons are particularly susceptible to such tragedies. Their training is long, their professional lives grueling and their earning span relatively brief. They are not provided, as a profession, with adequate pensions or retirement benefits. If surgeons hope to retire comfortably, to provide adequately for their dependents, they must make their earnings work for them. But surgeons have bought far more than their share of gilt-edged stock in barren mines and subaqueous real estate. Where, then, is security to be found? Find a competent advisor. Let him relieve us of most of the worry, and give us a better chance to realize a profit.

With ourselves and our families thus sketchily provided for, we must not forget that we have a civic responsibility, one which we must not shirk if our country is to remain as we want it. Pressed from all sides, we should find time, somehow, to serve when and where we can. The busiest man has obligations simply because he is one whose caliber is recognized and respected by his community. We have the further civic obligation, it seems to me, of taking an intelligent interest in politics. I confess that politics is a bramble patch in which I easily and often lose my way. I would therefore welcome a Physician's Political Digest, in which concise, coherent facts were divorced from the buncombe of the usual political verbiage.

Another obligation to our society, a duty which we alone can perform, relates to socialized medicine. This specter that has risen to haunt our colleagues abroad seems to be a growing threat to our own medical way of life

The idea is not new. It had its practical beginnings in the last century and reached complete development in the 1920's and 1930's in Soviet Russia and Nazi Germany. More ominously, almost every country on this side of the Iron Curtain has, since the end of World War II, significantly enlarged its program of compulsory medical care. France in 1945, Great Britain in 1946 and 1948, West Germany in 1949, and, less than two years ago, Sweden are only the most familiar examples.

From this brief history lesson we learn that socialized medicine can come suddenly or gradually, but that the trend has never been reversible. No nation has ever relinquished any of the compulsory medical benefits it has extended to its citizens.

What have we surgeons to expect and to fear from compulsory medical care? Let me give you a composite picture. Better still, let us imagine you as a figure in that picture. You are once again at the beginning of your medical career, this time under a system of socialized medicine. What kind of a job will you be permitted to do?

When the problem of your location has been settled for you, you begin to practice medicine. Your patients may or may not have chosen you. In Sweden, for example, they are assigned. In Germany, Holland or Britain, patients have a limited choice, but they must register with the physician for a stipulated period, ranging from three months to a year. Theoretically, patients may change doctors, and a doctor may even refuse a patient, but only if they are willing to undergo a vigorous red-tape battle with an entrenched bureaucracy.

You may be the more reluctant to refuse patients when you realize that your livelihood

derives from services rendered in bulk. Most systems of socialized medicine operate under some form of capitation fee. In England you would receive 18 shillings ($2.52) for every patient registered with you; in Holland, $1.10. Your income depends not on the number of consultations and visits, not on the amount of time and effort devoted to the individual case, not on the ingenuity of your art and science, not on the success or failure of your diagnosis and therapy, not on your patient's ability to pay, but on the number of names in your book.

A livable income will require that you register many more patients than can be attended by a doctor of average physical or mental endurance. Some doctors in England and Holland, for instance, register more than 4,000. You uneasily sense that you are scrambling not for patients but for customers. At any rate, you find that 3,000 or 4,000 customers—or rather, patients—add up to a pretty staggering work load: 100 cases in a work day, another patient every two or three minutes. A visiting American physician waited while a British colleague saw eighteen patients and filled out eighteen forms, in ninety minutes.

Under such conditions, what is it like to practice medicine? It has been described as "doctoring on the conveyor belt." As the years roll on, you are more and more apt to find yourself a glorified but overworked receptionist, a competent orderly. Your lack of facilities, your excessive work load and your fixed fee conspire to make you get rid of your patient and your responsibility for him as quickly as possible. You have become an allocating agent whose primary function is to send patients along to pharmacists, specialists, hospitals and sanatoria. The traditional doctor-patient relationship has been pretty well lost; in your professional world the term "family doctor" is taking its place in the vocabulary of a dead language. You can hardly be surprised if you find more and more of your colleagues comfortably content with the mediocrity of safe tenure. You can hardly be blamed if, in your mind and conscience, the sick man becomes the case, the "mechanical sum of individual and unconnected diseases," the name on the register, the name worth $2.50 a year.

Faced with such professional prospects in general practice, you may decide it would be wiser to become a specialist—a surgeon. You will probably become an employee of a governmentalized hospital. As such, in England, your salary for permanent full-time staff work begins at $3,900 and rises to about $7,000 at retirement (compulsory). (It may be more when the British physicians' current strike for higher pay is settled.) Whether a part-time or full-time surgical employee, you may find that your working conditions leave something to be desired. A patient has no choice of surgeon, but the surgeon has no choice either. The system which found the general practitioner getting rid of his patient as quickly as possible now catches up with you as a specialist. You may find yourself overloaded with patients who should never have been referred to you. You find less and less time to spend with the patients whose need for your services is greatest.

A final source of frustration remains. In every scheme of socialized medicine thus far devised there has been conflict between administration and profession. Only if you are lucky will your system be administered and supervised by physicians. Even so, you can anticipate close, restrictive, sometimes smothering regulation. You will have to become an expert in administrative procedure. The laws, regulations, official and semi-official announcements, to say nothing of their manifold technical interpretations, constitute a legal maze in which you must not let yourself be caught. As an adjunct of the Ministry of Health you may eventually conclude that professional skill is less important than a relish for bureaucratic existence and an aptitude for political infighting.

A final alternative remains. General practitioners and specialists in all free nations with compulsory medical schemes may supplement their labors and incomes by private practice, or they may disassociate themselves from the compulsory scheme altogether. There still are doctors with good private practices, but they are a diminishing minority in every country with socialized medicine or widespread, compulsory health insurance.

How can we avoid becoming faceless cogs in a state bureaucracy dedicated to medical inefficiency? We must point out to our neighbor, first of all, how much socialized medicine will hurt not only us but also him. We must warn him of the real meaning of such a system: that we could no longer fulfill our professional obligations as we know them. We must make sure he understands that when he is really sick he may have to wait his turn in a long line of

hypochondriacs and chronic freeloaders. We must demonstrate to his satisfaction that the cumbersome and extravagant machinery of bureaucracy will, in the long run, cost him more and give him less.

We can strike even more positive blows in the fight against socialized medicine by striking at the conditions which inspire people to want it. We can encourage and assist the establishment of more clinics, public and private, to provide necessary medical care for those who truly cannot afford it. We can encourage wider participation in non-monopolistic health insurance, particularly for that large group ineligible for charity benefits but unable to cope financially with an expensive operation or a long illness, so that under free, private enterprise no citizen is caught financially short.

Finally, what do we owe our profession? We must, of course, keep abreast of the latest developments and discoveries in surgery. As an expanding society legitimately demands more surgeons, we must strongly and steadfastly resist the easy solution—to relax professional standards. Instead we must answer that demand by encouraging more able young men to find careers in surgery. The most effective source of encouragement, I think, lies in our own experience. The things we found in surgery have been good: a stable and respected position in society; the opportunity to help our fellow men; the channeled curiosity that led and leads us to seek the unknown and know it. We must share the knowledge experience has been kind enough to give us. Wherever the opportunity presents itself in the life of the medical man, in the university lecture hall or the hospital corridor, not the least of his duties, and privileges, is to leave to posterity as much of value as is possible.

As I come to the end of these remarks, I realize I have presented more problems than solutions, problems impossible of complete solution. If I have done no more than make you aware of them, however, I am content; for I am confident that with growing wisdom those who have listened to me can reach far better solutions than I. Each of us can do only so much. Added together, our efforts may go far toward fulfilling the obligations that we, as surgeons, owe a free society.

WILLIAM J. NORRIS, M.D.,
Los Angeles, California

Investigation of Disturbances in Blood Flow Following Acute Anterior Poliomyelitis

A PRELIMINARY REPORT

HOWELL E. WIGGINS, M.D., FRANCIS E. WEST, M.D. AND THOMAS F. O'CONNELL, M.D.,
San Diego, California

DR. Charles W. Peabody of Detroit, Michigan, in 1930, became impressed with the incidence among post-polioparalytics of chilblains in severe winter weather and the difficulty in preventing discomfort from cold in the affected limbs. In the main, those were the cases in which growth was most significantly retarded. All such patients presented a markedly cyanotic, cold and clammy extremity on the cold days which was sufficiently in contrast to seem significant, in degree at least, with that of patients in whom retardation of growth was minimal.

There is clinical evidence that the circulation in a short extremity, weakened by poliomyelitis, is decreased as compared with that in the normal or less involved limb. This evidence, however, has been based upon the utilization of oscillometry and temperature change recordings.

Within recent years the development of the plethysmograph by Burch, Winsor et al. has made available to clinical investigators a method of accurately determining volume pulsation and volume blood flow. This is accomplished by utilization of a plastic cup sealed onto a measured volume of a digit (4 cc.); the air displacement effects of the digit's pulse are picked up by a pressure transducer, amplified electronically and are transcribed in graphic form. From this apparatus measurements of the amplitude of pulsation in the digit may be obtained as well as the wave form. In addition, relative blood flow through the part can be made by the venous occlusion method.

The technic used is as follows: Exactly 4 cc. of the digit is measured by immersing the digit into a 20 cc. vial of tincture of merthiolate® (of which 4 cc. has been removed) to the 20 cc. level. A digital plastic cup of the correct size is applied to the level of the "red line," and is sealed airtight with calking compound. A nondistensible plastic tube is attached to the digital cuff which is connected to the plethysmograph. A blood pressure cuff is placed at the wrist and inflated to approximately 25 mm. of mercury above the systolic pressure. The plethysmograph is then standardized so that there is a deflection of 10 cm. for 4 cc. of the part. Once the instrument has been standardized with the digit, the cuff at the wrist is inflated to approximately 70 per cent of the systolic and diastolic pressure. The rapid swelling of the part is transmitted through the plethysmograph onto the recorder and results in a rising baseline on the tracing. The relative blood flow is calculated by drawing a straight line along this baseline. The position of the line is determined usually by two points representing the beginning and ending of the first pulse wave which follows venous occlusion. The blood flow in the cubic millimeter of tissue per 4 cc. of part per second is determined by the number of millimeters the baseline rises in one second. By measuring the

TABLE I

Patient	Age (yr.)	Age at Onset of Polio (yr.)	Extremity Involved	Extent of Involvement	Circumference of Thigh (in.)		Circumference of Calf (in.)		Leg Lengths (in.)		Pulsation of Extremities						Pulsations of Digits: Toes (cu. mm.)				Blood Flow of Digits (cu. mm./sec.)				Skin Temperature of Digits (°c.)			
											Above Knee		Ankle		Below Knee		Before Heating		After Heating		Before Heating		After Heating		Before Heating		After Heating	
					Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
R. B.	11	5	Right	Moderate	12¾	14¾	8¾	11½	28½	29	8.0	13.0	6.0	8.0	7.0	14.0	.5	.5	2.5	1.5	.5	1.0	8.0	2.0	23.5	24.0	33.0	36.0
S. S.	10	6	Left	Moderate	13¼	11½	8¼	7⅞	28½	29	15.0 8.0	21.0 12.0	7.0 7.0	9.5 6.0	.... 12.0	.... 10.0	3.0 1.0	2.5 1.0	3.0 8.0	1.5 8.0	5.0 2.0	3.5 2.0	5.0 28.0	3.0 26.0	33.0 25.5	28.5 23.0	32.5 36.0	28.0 36.0
J. S.	7	3	Left	Severe	14½	10¼	10¼	7½	25½	24¼	14.0	9.0	11.0	5.0	....	....	3.0	.5	4.0	2.0	5.0	2.0	12.0	7.0	30.5	28.5	36.0	36.0
P. P.	7	4	Left	Moderate	13	12	9¼	6¼	23¾	23¾	13.0	8.0	6.0	7.0	11.0	8.0	2.0	2.0	2.0	2.0	2.0	2.0	5.0	6.0	28.5	30.0	35.5	36.0
M. L. B.	20	13	Right	Severe	16	19½	10⅛	12⅜	32⅞	33¼	7.0	9.0	5.0	8.0	6.0	9.0	1.5	1.5	6.0	7.0	2.0	2.0	9.0	8.0	28.0	27.5	33.5	35.0
C. A. B.	10	3½	Left	Severe	16¼	11⅜	11½	7½	30¾	29	13.0	6.0	5.0	3.0	11.0	5.0	1.0	1.0	8.0	6.5	1.0	1.0	23.0	18.0	25.5	24.5	35.5	35.0
E. McD.	13	6	Right	Moderate	14	16¾	8½	11½	32¼	33½	9.0	21.0	7.0	12.0	9.0	18.0	7.0	8.0	6.0	7.0	9.0	10.0	10.0	11.0	34.5	35.0	36.0	36.0
T. McH.	10	8¾	Left	Severe	14¾	10¾	10⅛	7¾	29¾	28¾	14.0 15.0	9.0 12.0	11.0 10.0	3.0 6.0	17.0 17.0	8.0 9.0	2.0 ...	1.0 ...	3.0	2.0 ...	4.0 ...	1.5	8.0 7.0	1.5 6.0	28.0 34.5	23.5 32.5	35.0 36.0	28.0 35.5
P. G.	7	1¾	Right	Severe	10¼	13	7	8½	22½	24¼	6.5	14.0	5.0	9.0	....	....	2.0	3.0	2.0	3.0	2.0	4.0	4.0	3.0	30.0	32.5	32.5	32.5
J. L.	11	7	Left	Severe	13½	11¾	10½	8½	30¼	29¼	11.0	8.0	8.0	6.0	16.0	7.0	1.0	1.0	4.0	2.0	1.0	1.0	6.0	9.0	23.0	23.5	35.5	36.0
R. P.	8½	4	Right	Severe	13	15	8	9	24	24	5.0	9.0	2.0	6.0	4.0	9.0	.5	.5	3.0	2.0	.5	1.0	4.5	4.0	24.5	24.5	35.5	36.0
M. M.	5	3½	Left	Moderate	11	8½	9¾	7¾	21½	21	11.0	11.0	5.0	5.0	5.5	5.5	1.5	1.0	1.0	3.0	6.0	4.0	13.0	9.0	33.5	31.5	36.5	36.0
L. K.	6	10 mo.	Right	Severe	9	11	6¾	9	21	22½	10.0	5.0	8.0	5.0	8.5	6.0	2.0	4.0	3.0	4.0	5.0	8.0	12.0	16.0	34.5	34.5	36.0	36.0
L. F.	8	4	Left	Severe	15¼	12¾	11	8¼	29	28½	13.0	7.0	9.0	4.0	13.0	8.0	1.0	1.0	7.0	5.0	1.0	1.0	10.0	8.0	26.5	24.5	36.5	35.5
J. E.	9	5	Left	Moderate	15¼	12	10¾	9½	26	25¼	11.0	9.0	6.0	4.0	12.0	8.0	1.5	1.0	2.0	2.0	6.0	10.0	10.0	13.0	28.5	32.5	34.5	36.0
D. N.	15	12	Left	Severe	17¼	12½	13½	10¼	34½	34	18.0	15.0	12.0	8.0	25.0	10.0	2.0	1.0	12.0	5.0	3.0	2.5	26.0	15.0	25.5	25.0	36.5	36.0
P. P.	17	15	Left	Severe	16	14	13	11	39	39	19.0	16.0	13.0	12.0	18.0	15.0	3.0	8.0	9.0	9.0	4.0	4.0	6.0	5.0	30.5	33.0	28.5	35.0
M. S.	11¾	9	Right	Moderate	15	16	10	11¼	30	30	10.0	13.0	6.0	8.0	11.0	12.0	.5	.5	6.0	5.0	0.0	.5	18.0	21.0	22.5	23.5	35.0	35.5

flow in the resting state and after artificially induced vasodilatation (general body heating, whiskey, drugs, nerve blocks), estimates of the potential blood flow can be deduced. This estimate of potential flow can be most helpful in making selections between fixed conditions of decreased circulation and those which are labile or alterable.

The utilization of a combination of standardized oscillometry, temperature recording and digital plethysmography in a study of the disturbances in blood flow following acute anterior poliomyelitis seemed a logical approach, and thus in 1955 a study of a group of eighteen postpoliomyelitis patients exhibiting alterations in circulation in the affected extremity was instituted. The results of this study form the basis of this preliminary report. (Table 1.)

The study was originally stimulated by our feelings that a correlation between the cold extremity and the shortened leg existed, and that with the advent of plethysmography, laboratory confirmation of this impression might be obtained.

Following the completion of this limited study it was at once apparent to us that our preliminary concept might not be substantiated. The most constant finding was in diminution of pulsations of the involved extremity which occurred in sixteen of the eighteen children. In the recording of the blood flow in the involved extremity, diminution occurred in only twelve of the eighteen children. Skin temperatures of the involved extremities were less than the uninvolved in eleven of the eighteen children tested. Body heating produced an increase in the pulsation of the digits of the involved extremity in fourteen of the eighteen children, increase in blood flow in seventeen of the eighteen children, and elevated the skin temperature in sixteen of the eighteen children tested. However, correlation between these expected findings of circulatory impairment and retardation of growth in the involved extremity was not consistent enough to allow valid conclusions.

It is our general impression that as the period of time from the onset of polio increases, stabilization of the vasomotor system progresses, thus explaining certain discrepancies in plethysmographic studies which also would assist in the explanation of the increment of shortening. The most profound circulatory changes that were noted, together with the rapid loss of growth in the involved extremity, occurred in a youngster whose onset of polio was of the most recent in origin. In this ten year old child 1 inch of shortening had developed in one and one-quarter years.

SUMMARY

Eighteen patients with postpoliomyelitic involvement confined primarily to one lower extremity were subjected to plethysmographic studies in an attempt to confirm a clinical impression of vascular impairment in the involved extremity. The circulatory studies were not all consistent with the amount of shortening of the involved extremity. The authors believe that these inconsistencies are probably related to the time differential between the onset of poliomyelitis and the initial study. We do not believe that our results warrant any conclusions with regard to therapy, but we are planning and would encourage a similar study of cases of more recent origin.

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DISCUSSION

FRANCIS J. COX (San Francisco, Calif.): The field of peripheral vascular surgery was developed on the theory that the blood supply to a given area was brought through normal afferent channels. An interchange of anabolic and catabolic products occurred in the peripheral vascular bed at capillary level. The veins carried the catabolic products away. If there was any local disturbance in this system, for example, arteriosclerosis, collateral circulation developed to solve the situation.

The biggest problem in understanding this mechanism lies in the relation between blood flow in muscle and flow to other tissues. We know how rapid blood flow can be out of a bone. This is proved by osteograms and more particularly with radioactive iodinated albumin. The radioactive material will disappear more rapidly from bone than it does from muscle tissue. If the blood volume flow from bone is that fast, blood must get into bone with equal rapidity, but as yet we cannot demonstrate the inflow into bone at all.

In a limb affected by poliomyelitis, removal of the normal sympathetic stimulus improves the circulation. Does this imply an overactivity of the sympathetic control, or is it pressure of the afferent side due to lack of muscle flow or bone flow?

The problem in poliomyelitis is a fundamental one to which no answer is as yet evident. Many lesions of the brain and central and peripheral nervous system produce changes in the peripheral vascular system which follow a similar pattern.

Any and all of the following lesions have the potentiality of producing signs of vascular insufficiency: lesions of the brain, for example, a stroke, can produce evident vascular change; lesions of the spinal cord, not solely confined to poliomyelitis; lesions of the nerve roots, for example, spina bifida; lesions of a peripheral nerve; and finally, a simple pain stimulus in the peripheral tissues.

One major block to statistical investigation of the problem is the known clinical observation that all of these patients do not show the same response in the vascular system as they do to their primary disease. We may see two essentially identical degrees of paralytic involvement in a leg: same age, same date of onset, etc. The first patient will have a warm painless extremity; the other, a cold, wet, painful cyanotic one.

The intermediolateral cell mass adjoins the anterior horn cells in the lumbar area. Perhaps this may have something to do directly with the involvement of the sympathetic nervous system in one patient and not in another.

The development and use of the plethysmograph, and especially the electronic plethysmograph, has added another valuable tool for investigators in the field of peripheral vascular disease. There is value indeed in the study of postpoliomyelitic paralysis and its concomitant vascular impairment. These findings should be correlated widely with other similar disease entities. If enough facts are accumulated, perhaps ultimately we can reach a better understanding of the vasomotor system generally.

This study of eighteen cases of postpolio paralysis is most interesting, but I hope the authors and others interested in the problem will expand their investigation into all similar disease entities, and most particularly into the field of reflex sympathetic dystrophy.

Dr. Rutherford Gilfillan of Children's Hospital in San Francisco is now working with a series of cases of poliomyelitis with involvement of the lower extremities, in which sympathectomy has been performed. The patients chosen for surgery were those who had a lowered blood flow, a lowered blood pressure and lowered skin temperature in the involved extremity.

Before surgery a reflex vasodilation test, skin temperature recordings at constant room temperature and plethysmographic blood pressures at the ankle level were made.

Following sympathectomy the blood pressures and blood flow on the involved side have returned to approximately normal levels. One adult with bilateral poliomyelitis was able to discard braces after operation. These same phenomena are also found in obliterative arterial disease as well as many other forms of neurogenic disease entities.

There is still a great deal of investigative work to be done.

FRANCIS E. WEST (closing): This problem of the inequality of limb lengths occurring after polio is a real challenge to the orthopedist, and has never been adequately answered.

As to the cause of the shortening, whether it is vascular or a loss of muscle mass, or associated with the disuse of the extremity, we believe, and have believed, that there is a need for positive information. We may need other forms of investigation to help us answer this problem.

I suspect that the problem will be raised in regard to the blood flow in the bone. We are interested in that, and perhaps from future investigations, by the use of isotopes or other methods, we will gain some valid information along that line.

We have presented our findings, which are very meager, only to stimulate further interest in investigation along this line.

We are facing the future with mixed emotions because we hoped and believed that we would get added information by studying fresh clinical cases shortly after onset of polio, and following them up for a period of several years. However, we hope that with adequate use of the Salk vaccine, perhaps the source of our fresh material will disappear. If not, we will continue the study and hope that others do the same.

Primary Subclavian Vein Occlusion

RALPH H. LOE, M.D., *Seattle, Washington*

THIS condition was first described by Paget [1] in 1875 and by Von Schroetter [2] in 1884, and hence has been called the Paget-Schroetter syndrome. It also goes under the name of primary, effort or spontaneous axillary or subclavian vein thrombosis. It is not to be confused with axillary vein thrombosis developing from infection or severe injury. This condition more commonly develops in the right arm of a young person following lifting or some muscular effort. Many cases have developed spontaneously with no apparent cause. While the onset may be rather sudden, it is more usual for the symptoms and signs to become manifest over the course of a few days. It is finally recognized by swelling of the arm and blueness of the skin, although a white or mottled arm has been described; with the arm abducted, a distended axillary vein may be seen or a cord-like, tender band felt if thrombosis exists. The complaints are usually vague and limited to discoloration, swelling and heaviness of the arm, axillary distress, pain in the shoulder, tingling and numbness of the arm and hand. There have been no reported deaths due to embolic phenomena and I have found but one case of lung infarction [25]. With proper treatment, serious disability is relatively short; however, in 75 per cent of cases there are persistent residual symptoms [3] which may take the form of distress in the axilla, a feeling of fullness and weakness in the arm, shoulder distress or pain at the base of the neck. Swelling of the arm may occur only with exercise, indicating an inadequate collateral circulation. There may be recurrent attacks resembling the original one.

ETIOLOGY

It is generally agreed that there is external venous obstruction occurring in the supra- or infraclavicular areas. The exact site of obstruction and mechanism has been a source of considerable debate. Veal and McFetridge [4] have stated that pressure from the head of the humerus on the subscapular muscle with the arm abducted may be the cause of venous occlusion. Wright [5] has expressed the opinion that the pectoralis minor muscle anchored to the coracoid process interferes with the axillary vein, particularly on abduction of the arm. McCleery et al. [6] believe pressure on the vein by the subclavius muscle and the costocoracoid ligament has an obstructing effect [28]. Narrowing of the space between the first rib and clavicle has been suggested as the cause by Loehr [7] and McLaughlin and Popma [8]. Hughes [9] does not believe that compression by the costocoracoid ligament is important, and described a prevenous phrenic nerve angling about the vein and made taut by deep breathing as a factor in the production of subclavian vein obstruction. Spasm and hypertrophy [6–8,27] of the anterior scalene muscle has been suggested. Pressure from the apex of the lung has been thought to play a part. I have used the term occlusion as I believe with others that an external obstruction is primary and, while thrombosis may occur, it is always secondary to the occlusion. Irritative venospasm may also be a factor in the production of cyanosis and swelling of the arm.

DIAGNOSIS

The diagnosis is usually obvious after a careful history is obtained and physical examination is made. In borderline cases various tests are available if required. There is decreased oxygen content of the venous blood; this was first noted by Horton [10] and confirmed by others [11,12]. The measurement of venous pressures in both arms is diagnostic, particularly following exercise [12] which will make the rise much more apparent on the obstructed side [13]. Circulation time [14] in the blocked side is decreased. Adson's [29] sign, a test for

scalenus anticus syndrome, should be looked for. There is a decrease or obliteration of the radial pulse when the patient rotates the head with chin elevated to the affected side while inspiring air. Venograms will show obstruction [*15*], usually in the region of the first rib, but it is impossible to tell the actual cause from the film. Collateral venous development can be demonstrated by infrared photographs of the upper torso.

The association of polycythemia has been reported in several instances; in one case reported by Stover and Herrel [*11*], early recognition and prompt venesection prevented a disastrous consequence. In another case the patient lost an arm [*16*].

TREATMENT

Immediate treatment may consist of sympathetic blocks as recommended by Ochsner and DeBakey [*17*], elevation of the arm and the use of anticoagulants. The nature of the early treatment does not appear to be important. Usually these patients can return to work relatively early although some symptoms may persist.

Matas [*18*] and Veal [*19*] have reported recurrent attacks, and others have reported continuing disability with symptoms previously described. It is in these patients that surgery may offer relief. McLaughlin and Popma [*8*], and Neij [*20*] were the first to report cases benefited by anterior scalinotomy. Thrombectomy [*21,22*], venolysis [*7*] and resection of a thrombosed axillary vein [*15*] have been reported with varying success.

CASE REPORTS

I wish to report two cases that occurred several years after radical mastectomy without evidence of metastasis.

Case 1. M. H., a sixty-eight year old woman, on whom I had performed right radical mastectomy for Paget's disease of the nipple in August, 1952, was seen on numerous occasions in follow-up. She was well except for soreness and numbness in her right arm and shoulder. In May, 1953, she reported considerable pain in her right upper arm. In March, 1955, she complained of more pain and swelling of the right arm. The distress became much more acute, and around Christmas, 1955, the arm became a very marked blue. There was no history of trauma. She was in Wyoming and was given heat and penicillin. The blueness gradually subsided but marked swelling of the right forearm and upper arm remained with considerable pain and distress in the axilla and hand. The patient was obese so that one could not actually identify the axillary vein; there was some tenderness in the supraclavicular area and in the axilla. X-ray of the chest and shoulder revealed no abnormalities. Examination of the right upper arm showed it to be 2 inches larger in circumference than the left. A venogram taken March 12, 1955, showed right axillary vein obstruction. As there was no evidence of recurrence of carcinoma, surgical exploration of the shoulder area was proposed. A combined incision hooking above and below the clavicle was made. The supraclavicular area was first explored. There seemed to be compression of the axillary vein as a finger above and below the clavicle showed marked narrowing of the space between the first rib and clavicle. The costocoracoid ligament was readily cut between the axillary vein and artery and anterior scalenotomy was performed. After this procedure there was a definite increase in the size of this space. The axillary vein was too deeply placed to visualize any change in its size.

One month after operation the diameter of the right arm had decreased 1 inch in size and there was a general decrease in all symptoms.

On January 4, 1956, the patient returned again with Paget's disease of the left breast. Left mastectomy was performed. The right arm was still moderately enlarged, but was much more comfortable than before operation. A follow-up letter on January 9, 1957, stated that the patient's condition was excellent; there was no further difficulty with the right arm.

Case 11. M. B., a sixty year old woman, was seen on June 4, 1955, with the chief complaint of swelling and blueness of the right arm. She had undergone radical mastectomy for cancer three years previously. No x-ray therapy had been given. One year previously some swelling in her right arm had been noted; six weeks previously she had experienced increased acute swelling and pain of the arm with tingling of the fingers. The arm became blue in color. When seen by us, her right arm measured 13 inches in comparison to the left arm which measured 10½ inches, and it was a mottled blue in color. The radial pulse in the right arm was weaker than that in the left. Palpation of the shoulder girdle indicated a narrower space below the clavicle on the right than on the left. There was moderate edema of the right arm with obvious increased tension of the axillary vein. Chest x-rays were negative for metastasis. The patient refused treatment but was seen in a week with improvement. She was told that her condition should gradually improve and that if symptoms persisted an operation might be considered. She has not returned for follow-up.

Comment. Swelling of the arm due to lymph blockage following radical breast surgery is relatively frequent, but acute venous occlusion of a non-metastatic nature has seldom been reported. It would appear that following radical mastectomy the spacial relationships of the structures passing from the supraclavicular area beneath the clavicle and into the upper arm could be sufficiently altered by severance of the pectoralis minor and major muscles to increase the incidence of shoulder syndrome or subclavian vein occlusion.

Delayed swelling of the arm has usually been considered due to metastasis. However, in view of the anatomic changes possible, there ought to be a greater incidence of swelling due to later occlusion of the subclavian vein of a non-malignant nature. These cases deserve an inquiry into symptoms of a shoulder syndrome or a history of cyanosis of the arm. A careful examination for the presence of increased venous pressure is indicated. Wyburn-Mason [*27*] noted that four of thirty-two patients with edema of the arm after radical mastectomy had elevated venous pressure; marked dropping of the shoulder was present in these. Elevation by a sling reduced the edema. He suggested that removal of a portion of the first rib might relieve the condition. Macdonald [*26*] reports resection of the axillary vein proximal to the entrance of the cephalic vein without producing swelling in the arm. Therefore, any swelling of the arm due to occlusion would have to be above the entrance of the cephalic vein.

Case III. S. S., a twenty-nine year old man, was first seen on March 21, 1950, with the chief complaint of swelling of the left arm beginning seven days previously following the lifting of heavy plates of glass in his occupation as a glazier. During the course of the week he noted an increasing enlargement of the arm as well as a bluish discoloration. There was stiffness on opening and closing the hand as well as decreased strength. Examination of the abducted arm showed that the axillary vein was markedly distended. The arm was bluish in color and was considerably larger than the right arm even though the patient was right-handed. The left arm measured approximately ¾ inch larger than the right arm. An x-ray of the chest and an electrocardiogram revealed no abnormalities. He was treated by sympathetic block, elevation and dicumarol. At the time of discharge from the hospital at the end of five days the arm was considerably better and the bluish discoloration was less noticeable. He was back at work in about three weeks. However, some symptoms persisted in the form of slight swelling and weakness of the arm, fullness and pressure under the axilla, and distress in the shoulder and base of the neck of a vague nature. A venogram taken on January 15, 1951, showed a very definite block in the area of the first rib. Surgical exploration was suggested but was refused. A venogram in 1954 showed the same block. (Fig. 1.) The symptoms persisted and finally became so annoying that the patient consented to surgical exploration on January 17, 1956, approximately six years after the onset of his trouble.

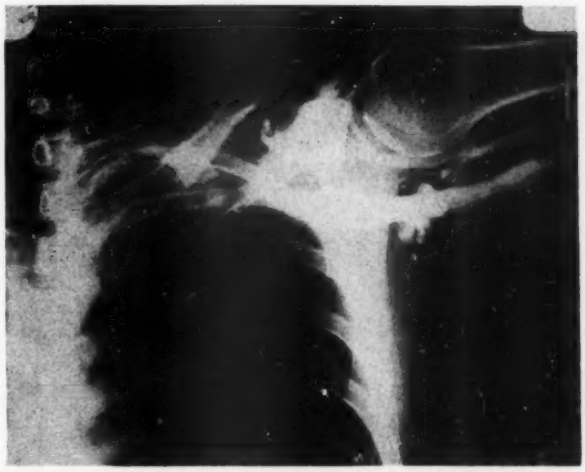

Fig. 1. Case III. Venogram taken in 1954 showing subclavian vein obstruction and collateral circulation which had developed during the past four years.

The proximal portion of the 4 inch incision was made above the clavicle and the distal portion below. The incision easily exposed both the supraclavicular and infraclavicular spaces, permitting complete inspection of the axillary and subclavian vein and the tissues which might be obstructing these vessels. A clip was placed on the lateral border of the anterior scalene muscle above the clavicle and on the edge of the costocoracoid ligament at the medial side of the vein. A venogram was taken after injecting 10 cc. of 30 per cent diodrast® through a polyethylene tube which had been inserted within 3 inches of the axilla through the median basilic vein. In this picture one cannot tell whether the block is at the subclavius muscle or the anterior scalene muscle. The costocoracoid ligament was cut between the axillary vein and artery and another venogram was made. (Fig. 2.) No improvement occurred. A markedly hypertrophied anterior scalene muscle was now more carefully isolated and the subclavian vein was seen tightly stretched and flattened over its anterior aspect. The anterior scaline muscle was then cut and some filling of this vein was seen to take place. A venogram was again made using 70 per cent

FIG. 2. Case III. Operative venogram with 30 per cent diodrast after cutting the costocoracoid ligament which is indicated by the lower clip on its inner border. The upper clip is on the lateral border of the anterior scalene muscle. The obstruction has not been relieved.

urokolon. The subclavian vein can be seen to have opened, and the deformity produced by the hypertrophied anterior scalene muscle is clearly visible. (Fig. 3.) This deformity seemed to be exaggerated by the fulcrum effect of the costocoracoid ligament. (Fig. 4.)

The patient was discharged from the hospital in four days with an immediate improvement in his symptoms. Follow-up examination five weeks after the operation indicated that he was performing his full duty as a glazier. He stated that he had much more freedom of motion in his left arm, the axillary fullness had disappeared as well as the supraclavicular discomfort, the congested feeling in the arm was gone, and he could now comfortably carry on the lifting of heavy plates of glass. There was still slight distention of the axillary vein on inspection. When seen again on November 5, 1956, he had no complaints referable to the arm. There was still slight swelling. A venogram taken on January 12, 1957, was about the same as the last seen except that the axillary vein was about one-half its previous size.

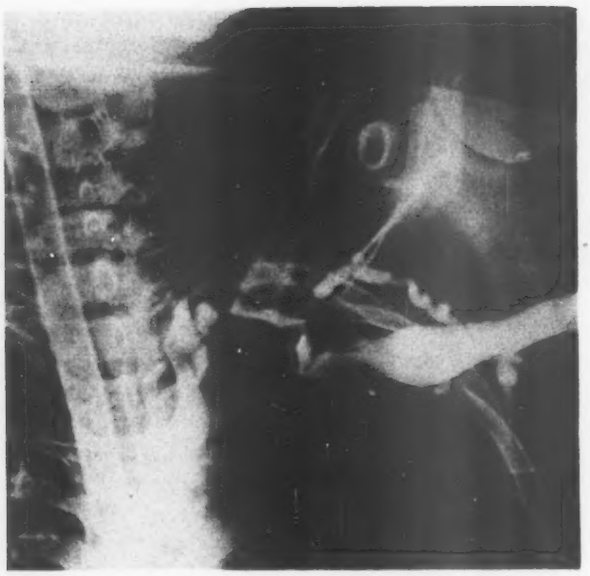

FIG. 3. Case III. Operative venogram with 10 cc. of 70 per cent urokolon after anterior scalenotomy. The subclavian vein, now open, shows very clearly the distortion produced by the hypertrophied scalene muscle. The remaining clip is on the costocoracoid ligament.

COMMENTS

It would appear from the literature that spontaneous axillary or subclavian vein occlusion represents a complete closure of a vein which may have been previously partially obstructed by external pressure in either the supraclavicular or infraclavicular areas. Venograms have usually indicated a block as the vein crosses the first rib. The venogram in patient S. S. (Case III) very definitely indicated that cutting the anterior scalene muscle opened up the subclavian vein. The costocoracoid ligament might have played the role of a fulcrum in angulating the vein, but its severance did not release the vein. The trigger mechanism might be the muscular effort so commonly associated with the beginning of this condition, resulting in either a hemorrhage or a spasm of the anterior scalene muscle. Pressure of the apex of the lung behind the muscle in straining may aid in narrowing the space. The scalenus anticus hypertrophy probably resulted from the patient's occupation which necessitated lifting heavy plate glass while bending the head backwards or sideways.

Actual thrombosis [*21,22*] of the axillary vein has been described and removed by surgeons operating during the acute phase. The results were satisfactory in a few cases. In several

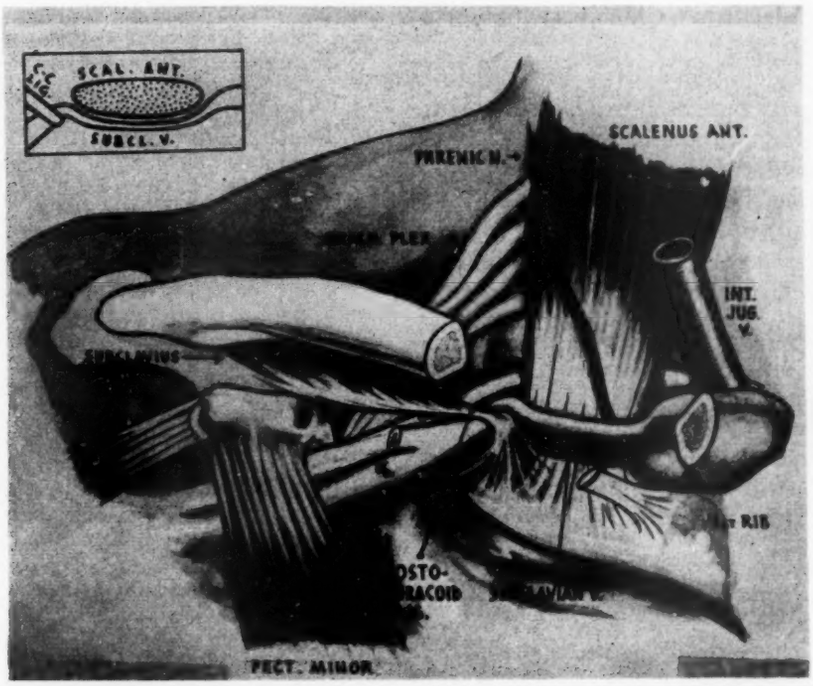

FIG. 4. Diagrammatic sketch taken from the patient in Case III, with a portion of the clavicle removed for clarity of demonstration. The scalenus anticus muscle was markedly hypertrophied, completely obstructing the subclavian vein. The insert shows our conception of the costocoracoid fulcrum effect in this case. The vein showed no evidence of previous thrombosis.

instances [*15*] there has been no thrombosis found at surgery. In neither of our delayed operative cases could one determine that thrombosis had ever occurred. Others [*5*] exploring the same area have had the same experience.

The fact that there has been but one embolism reported in several hundred cases strongly suggests a pre-existing external occlusion that would prevent a thrombosis from breaking off.

The prompt treatment of polycythemia vera complicating one of the reported cases undoubtedly played a part in saving an arm.

The treatment of the acute phase should be conservative, as previously described. Early return to activity is not dangerous, but may be prevented by recurrent swelling or pain in the arm.

A rather large percentage of patients have residual symptoms in the shoulder and arm which greatly resemble those generally associated with the scalenius anticus syndrome. It would appear that this condition is actually another manifestation of that syndrome. The venogram and results in Case III would support this conclusion. There are at least ten other cases in which relief of symptoms [*6,8,20,23,24,30*] was obtained by anterior scalinotomy. No other operative procedures have given such good results. Wyburn-Mason's [*27*] patient had symptoms of brachial arterial and venous pressure, and was relieved by anterior scalenotomy plus partial resection of the first rib. While severance of the anterior scalene muscle may be all that is required in most cases, I still believe that the incision should be made in such a manner as to expose the supra- and infraclavicular spaces to be sure that all pressure points have been released. Resection [*27*] of a portion of the first rib may be necessary in rare cases. Operative venograms can be of help in determining the precise surgery required as well as the results of such surgery.

SUMMARY

1. Three cases of acute subclavian vein occlusion are reported. Two developed in patients previously operated upon for carcinoma, but without evidence of metastasis. Changes in anatomic relationships resulting from cutting the pectoralis major and minor muscles may

predispose the patient to venous occlusion or a shoulder syndrome.

2. Anterior scalenotomy with the possible aid of severance of the costocoracoid ligament relieved residual symptoms in two cases.

3. An operative venogram with silver clips appropriately placed clearly demonstrated that cutting an enlarged anterior scalene muscle released the pressure on the subclavian vein.

4. Acute subclavian vein occlusion may be considered the venous component of the scalenus anticus syndrome.

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DISCUSSION

Dean K. Crystal (Seattle, Wash.): Dr. Loe has called attention to an interesting condition which can prove to be genuinely serious.

There are not very many examples of effort syndrome in the shoulder associated with spontaneous thrombosis of the subclavian and axillary vein, but those which do occur can result in a form of disability which is very troublesome to the patient and often extremely annoying to his attending surgeon. In my experience there is little threat to the patient's life from this lesion, but there are many reasons to limit the morbidity as much as possible.

It is important that the vise-like action of a number of the normal musculoskeletal structures in the lower neck be appreciated by anyone who is called upon to treat this condition. I agree with Dr. Loe that removal of the subclavius muscle constitutes an excellent adjunct to scalenotomy

and I would agree with him also that it does not unnecessarily complicate the procedure.

I think that prompt therapy can be urged upon all who treat this disorder because I believe that in addition to the benefits of dicumarol or some other anticoagulant substance and proper positioning of the limb, an apparent interest in the patient's problem can do a great deal toward achieving a salutary attitude on the part of the patient. It has been my observation that a number of people with this syndrome have been told by their doctors that there is very little wrong with them and that they really have nothing to worry about. Such an approach almost always engenders a sort of resentment which makes for trouble later even in the form of litigation.

That brings me to my final point on this matter. The morbidity is sufficiently prolonged so that it may be extremely difficult to assess disability properly in people who have the effort syndrome involving subclavian or axillary vein thrombosis. When an accident occurring at work has led to the trouble it may be very difficult to get a patient to register any improvement whatever in the condition of his arm. He may feel that the settlement proposed has been inadequate or that the examining doctors are not sufficiently sympathetic toward his distress, and for that reason he may be a thoroughly resentful person. Prompt recognition and systematic therapy along with operative exposure, if the condition seems to warrant it, will do a great deal toward minimizing not only the duration of morbidity in this condition but also the patient's attitude toward it.

SIEGFRIED F. HERRMANN (Tacoma, Wash.): I have enjoyed this presentation very much, particularly because it reminds me of a horrible experience that I had many years ago.

I was just beginning in general practice in a little town in Minnesota, when I was called one day to see a woman who lived only three blocks from our house, who had suffered an injury.

She had been working on the outside of the house with a ladder, washing the upstairs windows. The ladder slipped and she caught it as it fell. She was standing on the ground.

She noted an immediate pain in her left arm and up in the left axillary region. It was so severe that she wanted a doctor.

She was a big, strong, husky woman, easily able to catch a ladder without getting hurt, one would think; but here she was, complaining of very severe pain, and I had no idea what was wrong with her. I watched her arm turn blue, and then swell.

She had pulses in both radial arteries. I gave her some morphine. I did not know what to do and said I did not know what she had, so another doctor was called from the little neighboring town. Dr. Butts and I wondered what we could do for this woman, any more than giving her morphine, as we watched the arm get bluer and more swollen.

Her pulse began to get weaker and we resorted to adrenalin, although we did not know what good that would do.

Within two hours she was dead.

I did not know what had happened, but not long after in the *J. A. M. A.* (March 15, 1924) there was an article which described this condition: injury to the axillary or subclavian vein. It described the influence in some people of an abnormally placed costocoracoid ligament, or some band of fascia that might injure the vein.

Perhaps there were some fatalities mentioned in the article; I have never forgotten mine. I do not know what I could have done about it, but there must have been some anatomic reason why the sudden jolt injured the subclavian vein, and it must have been high because the whole arm was involved. Then, I suppose, a propagating thrombus and a massive pulmonary embolus formed which caused her death.

I am mentioning this case only because these episodes can be quite acute.

IAN MACDONALD (Los Angeles, Calif.): There have been periodic articles concerning the importance of an alleged axillary venous obstruction as the causative mechanism for postmastectomy lymphedema.

I should like to say only one word of caution here against the interpretation of this situation on the basis of a single film, or films taken without the aid of an automatic cassette changer.

Some two years ago we obtained venograms on fourteen women following radical mastectomy. Eight of them had lymphedema of significant degree; six of them had no lymphedema.

Of the fourteen, there were only three in whom we could demonstrate a genuine axillary or subclavian venous obstruction on the basis of venograms. However, almost half of the group did show spasms of the axillary vein, and only by taking serial venograms at intervals of $\frac{1}{100}$ second, in some of these women, could one demonstrate that the apparent axillary venous obstruction was spasm and not genuine mechanical obstruction.

RALPH D. CRESSMAN (Palo Alto, Calif.): I am interested to hear Dr. Loe speak about this condition.

You are all aware that it always bring to mind the case you had, and I have seen this situation in a young college student whose difficulty followed the playing of handball. He had not only an acute episode but also a relapse.

I wonder why Dr. Loe and Dr. Crystal are so conservative in their approach? They have pointed out that some 75 per cent of these patients do have residual symptoms. Therefore, I wonder what

should delay us in exploring the vein, and if this is due to actual obstruction, why it should not be relieved immediately.

I did not perform exploration on my patient because the older literature does not mention the possibility of doing this.

I wonder if Dr. Loe would like to comment on why we should not immediately explore the vein and relieve the obstruction.

RALPH H. LOE (closing): I would like to say first that no one has had very much experience with this condition. Dr. Hughes collected 320 cases from the literature in 1950, and the most that any one person had reported from his own experience, I think, was five cases.

In approximately 400 reported cases, I was not able to find any with a genuine acute fatality from a thrombus originating from a spontaneous axillary or subclavian vein occlusion. I think that Dr. Herrman should report his case.

The reason, of course, that you do not see fatal cases is apparently because of the proximal obstruction in the subclavian vein, which prevents, in the large majority of cases, a thrombus breaking loose.

I was interested in Dr. Macdonald's comments. I think that one has to be very cautious in suggesting that postmastectomy swelling may be of venous origin. But, as he has indicated, I think there are a few cases, and I am quite aware that the venograms are difficult to interpret. I think his idea of taking films in rapid succession is an excellent one.

I think the other tests, such as venous oxygen content, circulation time and venous pressures, are important in arriving at the diagnosis of this condition. I believe that there are probably a certain number of patients, especially with delayed swelling of the arm following radical mastectomy, in whom the swelling is of venous origin; granted that most of them are of just the ordinary lymphatic type of blockage.

Regarding Dr. Cressman's comments on immediate surgery, I am not sufficiently sure of the patients who will and will not have a thrombus to be willing to operate immediately. Most patients recover without surgery and that is the reason that I would not particularly urge it unless the symptoms persisted.

Distal Operative Arteriography—An Aid in Peripheral Arterial Surgery

WILTON A. DOANE, M.D., *Santa Barbara, California*

RAPID advances in technics for correction of peripheral arterial occlusive disease have made it possible to salvage many patients who previously would have been considered completely incurable or relegated to sympathetic ganglionectomy and medical management.

The over-all results of such new technics have been amazingly good in many series, yet a varying percentage of immediate and late failures has occurred. Failures in the direct surgical attack on obstruction of peripheral arteries may be due to (1) technical errors in endarterectomy or anastomosis or (2) improper selection of cases. The primary concern of this paper is to improve the selection of cases for direct arterial surgery by dispelling any doubt about the adequacy of arterial circulation distal to a localized arteriosclerotic occlusion. Rapid multiple exposure films of the progress of dye through an extremity are usually diagnostic, but this method is not universally available. Single peripheral films taken a few seconds after aortography or peripheral arteriography may fail to visualize the radiopaque dye at the proper instant to show both the proximal and distal extent of the block and the condition of the arterial tree distal to the block.

Distal operative arteriography was advocated by Barker [1] in 1954 to provide information about the arterial tree distal to a block which is often not obtained by a proximal arteriogram. Distal arteriography should enable one to demonstrate the exact location of the occlusion, the size and condition of the vessel distal to the occlusion and the adequacy of the outflow tract. Failure to visualize a patent arterial lumen at or proximal to the popliteal bifurcation constitutes a contraindication to direct surgical procedures for relief of segmental occlusion since the rate of failures of anastomoses rises sharply in the smaller vessels distal to this point. Significant narrowing or occlusion of the arterial tree distal to the popliteal bifurcation represents another contraindication to direct surgery.

Crawford et al. [2], in reporting their experience in surgical correction of arteriosclerotic occlusive disease, listed failures in three of twenty-four femoral by-pass grafts and two of twelve femoral blocks in which excision and grafts were utilized.

In analyzing their few failures, they attributed all of them to an inadequate peripheral outflow bed and believed furthermore that the failures were the result of improper selection of cases.

Oudot and Beaconsfield [3] took bilateral direct femoral arteriograms several days prior to aortic resection for aortoiliac thrombosis. They believe that demonstration of patency of the arterial tree below the occlusion is one of three major requisites for a successful result.

Julian and associates [4] classified their cases of peripheral obstruction into four groups on the basis of arteriographic findings. The first two groups, both suitable for direct surgical attack, presented a demonstrable area of occlusion of variable length with evidence of a patent distal lumen. The fourth group showed diffuse narrowing of the major arterial trunk with a barely patent lumen throughout. These they deemed unsuitable for direct surgery of any type. The third group, however, was characterized by an abrupt obstruction and adequate collaterals, but no visible distal arterial trunks. At operation in this group of cases they found several instances of "a surprisingly good channel distal to the obstruction."

Distal operative arteriography is applicable to obstruction anywhere between the aortic and popliteal bifurcations. In iliac and proximal

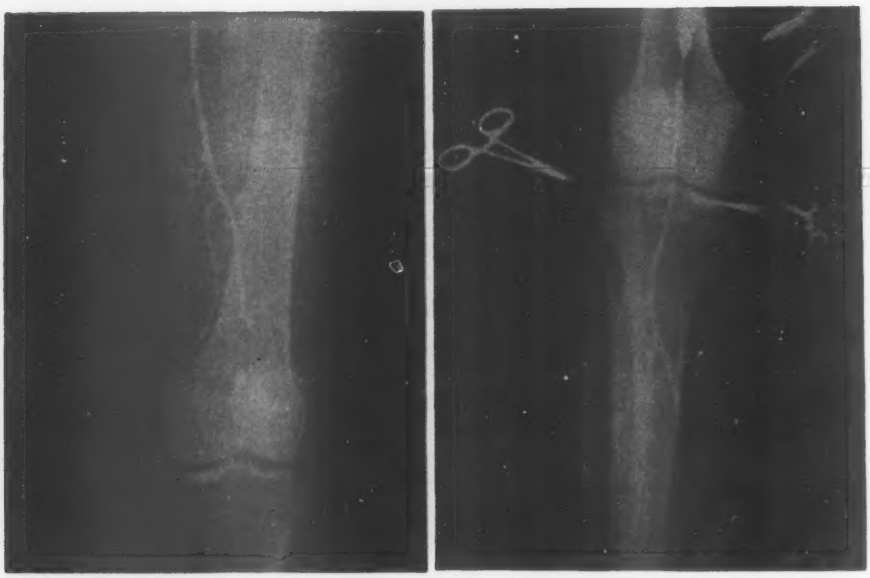

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FIG. 1. Direct femoral arteriogram visualizing localized block in femoropopliteal artery but failing to visualize popliteal bifurcation or arterial branches distal to the bifurcation.

FIG. 2. Distal operative arteriogram showing complete visualization of the popliteal bifurcation and distal branches.

femoral artery occlusion, the patient may best be managed in a supine position. However, in occlusions in the distal femoral or proximal popliteal artery, simultaneous exposure of the femoral and the popliteal bifurcation may be difficult. The prone position with the patient turned on the side to be operated upon and the opposite thigh acutely flexed out of the field was suggested by Graham [5] and has given unusually good simultaneous access to these two areas.

Utilization of this procedure as a first step in direct arterial surgery obviates the necessity for earlier distal arteriography as a separate procedure. Furthermore, preoperative arteriography invariably produces edema and ecchymosis about the site of injection which increases the technical difficulties in exposure and vascular anastomosis in the seven- to ten-day period following arteriography. However, in performing vascular by-pass, replacement or endarterectomy immediately following operative arteriography, one finds minimal technical difficulties due to tissue reaction.

TECHNIC

The distal artery is exposed through a short longitudinal incision and a tape passed about the artery proximally for easy handling during dye injection. After placing the cassette and positioning the portable x-ray machine, a No. 19 needle is inserted just below the obstructed area and 10 to 20 cc. of dye are injected rapidly. If visualization of the distal outflow tract is especially desired, proximal occlusion of the artery by pinching the tape aids in increasing the radiopacity of the artery. We have not used preliminary intra-arterial heparin as advocated by Barker [1], but have used small doses of 1 per cent procaine and heparin in the artery following arteriograms. Trauma to the artery may be reduced by using a short plastic tube to connect the syringe to the needle. The film exposure is made just before completion of dye injection when 1 or 2 cc. remain in the syringe. Any arterial bleeding at the removal of the needle is readily controlled by massaging the injection site with a dry sponge. Sometimes it is necessary to expose the artery at several levels before finding patent artery for distal injection. After studying the resultant arteriograms, one may either proceed with direct arterial surgery or simply close the wound.

CASE REPORT

A fifty-three year old white man, a sheet metal worker, was first seen in June, 1956, complaining

of weakness and numbness in the left leg and foot after walking less than one block. He also noted cramps in the left calf muscles and coolness of the left foot. General physical examination disclosed normal femoral pulsations on each side, normal popliteal and distal pulsations on the right side, but absent popliteal and distal pulsations on the left side. He presented a delayed capillary refilling on the left side, and the left foot and leg were cooler and paler than the right. Direct femoral arteriogram on July 25, 1956, revealed a block in the upper popliteal artery approximately 3 cm. long. The lumen of the popliteal artery was visible on the femoral arteriogram but no information was available on the films regarding the popliteal bifurcation and the distal branches of the bifurcation. (Fig. 1.) Therefore, on August 16, 1956, with the patient in the semi-prone position, the popliteal vessels were exposed through an S-shaped incision across the popliteal space with an extension up the medial aspect of the thigh along the distal femoral vessels. The site of obstruction was exposed, and a distal operative arteriogram revealed a normal popliteal bifurcation with good filling of the posterior tibial, anterior tibial and peroneal branches distal to the bifurcation. (Fig. 2.) The area of popliteal occlusion was densely adherent to the surrounding structures and in an attempt to salvage all possible collaterals, a short segment of saphenous vein was used as a by-pass about the area of occlusion. Postoperatively, the patient presented a full bounding popliteal pulse but no pedal pulses were ever felt. His exercise tolerance, which previously was limited to one-half block, was greatly increased so that he was able to walk eight to ten blocks without any claudication or weakness.

SUMMARY

In summary, the case for distal operative arteriography has been presented with a brief discussion of the technic utilized and description of an illustrative case. This diagnostic procedure offers an opportunity for more accurate selection of cases in evaluating arteriosclerotic occlusion for direct arterial surgery.

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DISCUSSION

Edwin J. Wylie (San Francisco, Calif.): Any arteriographic means of demonstrating the distal arterial tree prior to surgical attack of a more proximal diseased segment deserves full consideration. Our experience, however, has led us to advocate the use of serial exposure of extra-long films with proximal aortography to accomplish this end, in preference to the method outlined by Dr. Doane. In our method, 36 inch films are passed by hand through a plywood tunnel with built-in grids. Exposure of four films over a ten-second interval (the first exposure is made at the completion of the dye injection) results in visualization of the proximal and distal tree in one study.

The publicity given to rare complications of aortography has created unjustified concern about the use of this diagnostic method. In a series of 620 aortograms at the University of California Hospital, only one fatality occurred and this was due to renal artery thrombosis. One patient sustained spinal cord damage but recovered almost completely. Both of these complications followed injection of the upper abdominal aorta for the demonstration of lesions high in the aorta. We have had no difficulty following the injection of the lower abdominal aorta in patients with distal iliac and femoral artery lesions.

We believe the advantages of this method outweigh its slightly greater risk. By using this procedure in numerous cases of femoral artery thrombosis, we have been able to demonstrate unsuspected preocclusive lesions at other sites, such as the proximal iliac arteries or the opposite extremity.

These findings have resulted in alteration or extension of our surgical approach, and when the primary lesion is of questionable operability, such findings have been the deciding factor against any surgical intervention, other than perhaps sympathectomy alone. Any benefits of a locally successful grafting procedure or thromboendarterectomy often may be nullified weeks or months later if an occlusion develops at a higher site or in the opposite extremity.

As Dr. Doane has indicated, operability of a femoral lesion is determined largely by the condition of the arterial tree distal to the block. We have adopted fairly strict criteria of operability for this type of lesion. The distal femoral or popliteal artery must show a wide lumen free of distal obstruction or significant narrowing. Only one-third of the patients studied radiographically have satisfied these requirements. Unfruitful exploratory operations have thus been avoided in two-thirds of these

patients by using this method rather than open distal arteriography. In patients whose legs are of questionable viability, operative incisions about the knee can be avoided, and thus a potential operative site for amputation is not compromised.

A further disadvantage of the method of open distal arteriography is the difficulty of selecting the best site for arterial exposure. In some cases the lower level of complete occlusion is at the distal end of the adductor canal. The artery may be sclerotic, however, as far as the lower popliteal level, indicating a preferable graft site below the knee joint. In such a circumstance either multiple incisions or an unnecessarily wide exposure is required after preliminary exposure of the lower femoral artery.

JACK A. CANNON (Los Angeles, Calif.): I agree with Dr. Doane with regard to this subject. Nevertheless, I admire wholeheartedly the very beautiful films that Dr. Wylie is able to get. I think that the problem of expense and the team required in order to obtain films by his technic is insurmountable in many places. We are still stumbling over the problem of obtaining four 36-inch film cassettes and the expensive grid screens that go with them. Consequently, we have continued to use distal operative arteriography for this problem. We have found to our surprise that frequently a distal arterial tree can be shown to be patent even though at translumbar, one-exposure aortography, such did not appear to be the case. In addition, we find to an increasing extent that a very accurate appraisal of the status of the distal tree is possible on clinical appraisal alone.

However, I do believe that if Dr. Wylie's technic is used, i.e., multiple exposure aortography, it is apparent that the distal tree can be evaluated with great accuracy.

J. KARL POPPE (Portland, Ore.): I would like to compliment Dr. Doane on his excellent illustrations and stress the advantages of obtaining as much information as possible before the day scheduled for the arterial transplant. Additional films of the full length of the lower extremities taken a few seconds after the abdominal aortogram have showed satisfactory reconcentration of the radiopaque material in the extremities distal to the iliac obstruction.

I have been pleasantly surprised to note satisfactory results with iliac grafts in patients with short popliteal artery occlusions when concomitant sympathectomy is performed with the arterial replacement.

WILTON A. DOANE (closing): I quite agree with Dr. Wylie that aortography is indicated if there is any question about the femoral pulsation. In view of reported complications following aortography, I prefer not to use this diagnostic technic if the femoral pulsations appear to be perfectly normal.

I, too, wish to commend him on his films, and believe that this technic is particularly satisfactory in his hands.

The popliteal bifurcation would be my choice as to the site of distal injection whenever there are several areas of partial narrowing of the arterial tree above that site. I believe that distal operative arteriography is an accurate, certain method of diagnosis, especially in instances in which the distal arterial tree is not well delineated by any other method.

The Bypass Graft

ITS USE IN ARTERIAL OCCLUSIVE DISEASE

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Arteriosclerotic arterial insufficiency of the peripheral blood vessels is often a result of a localized obstruction with a patent arterial system above and below the obstructing lesion. Resection of the diseased segment and insertion of a suitable graft [4], thromboendarterectomy [1,2,8,18,19] and the bypass graft [3,4,9,10,15] have all been used with a varying degree of success in the treatment of peripheral arterial obstructions.

The purposes of the present study are: (1) to determine the value of the homograft and the bypass technic in the treatment of segmental obstructions of the iliac, femoral and popliteal arteries; (2) to illustrate the value of the vasographic peripheral vascular laboratory examination as an aid in selecting patients who should benefit by surgical therapy; and (3) to provide objective evidence concerning the success or failure of the bypass graft in improving the circulation to the extremities. This report is based upon the evaluation of our experience with the lyophilized homograft used to bypass localized arteriosclerotic obstructions in the arteries of the legs.

MATERIAL AND METHODS

Data concerning fifteen patients are included in this study. (Table I.) Twelve patients were men and three were women. Twelve were Caucasians and three were Negroes. The ages varied from thirty-nine to seventy-six years, with the majority falling into the fifth decade. Obesity was rarely encountered. Twenty-three bypass homografts were used in the fifteen patients. The right leg was involved in eleven instances and the left leg in twelve.

The obstructions which were bypassed were located in the following arteries: the superficial femoral in thirteen cases, the external iliac in twelve, the common iliac in eleven, the internal iliac in ten, the common femoral in eight, the aorta in three, the popliteal in two, and in the arteries distal to the popliteal in two. In each instance lyophilized arterial homografts were used for the bypass. These were obtained from the Los Angeles County Heart Association Artery Bank and were prepared according to the technic of Hufnagel [6]. The arterial segments utilized included external and internal iliac arteries and small abdominal and thoracic aortas. Femoral vessels were not available. If the segment to be bypassed was long, several pieces of homografts were sutured together. The longest graft made—34.5 cm. in length—consisted of three segments and extended from the lower abdominal aorta to the popliteal. (Fig. 1C.)

Table I

Patients (15):	
Men	12
Women	3
Bypass homografts (23):	
Right leg	11
Left leg	12

In addition to the bypass graft, twenty-three additional surgical procedures were performed. (Table II.) Eight bilateral lumbar sympathectomies were accomplished in a manner which has been described [12]. The five thromboendarterectomies were confined to the common iliac arteries. Two thrombectomies were performed; one clot was evacuated from a graft

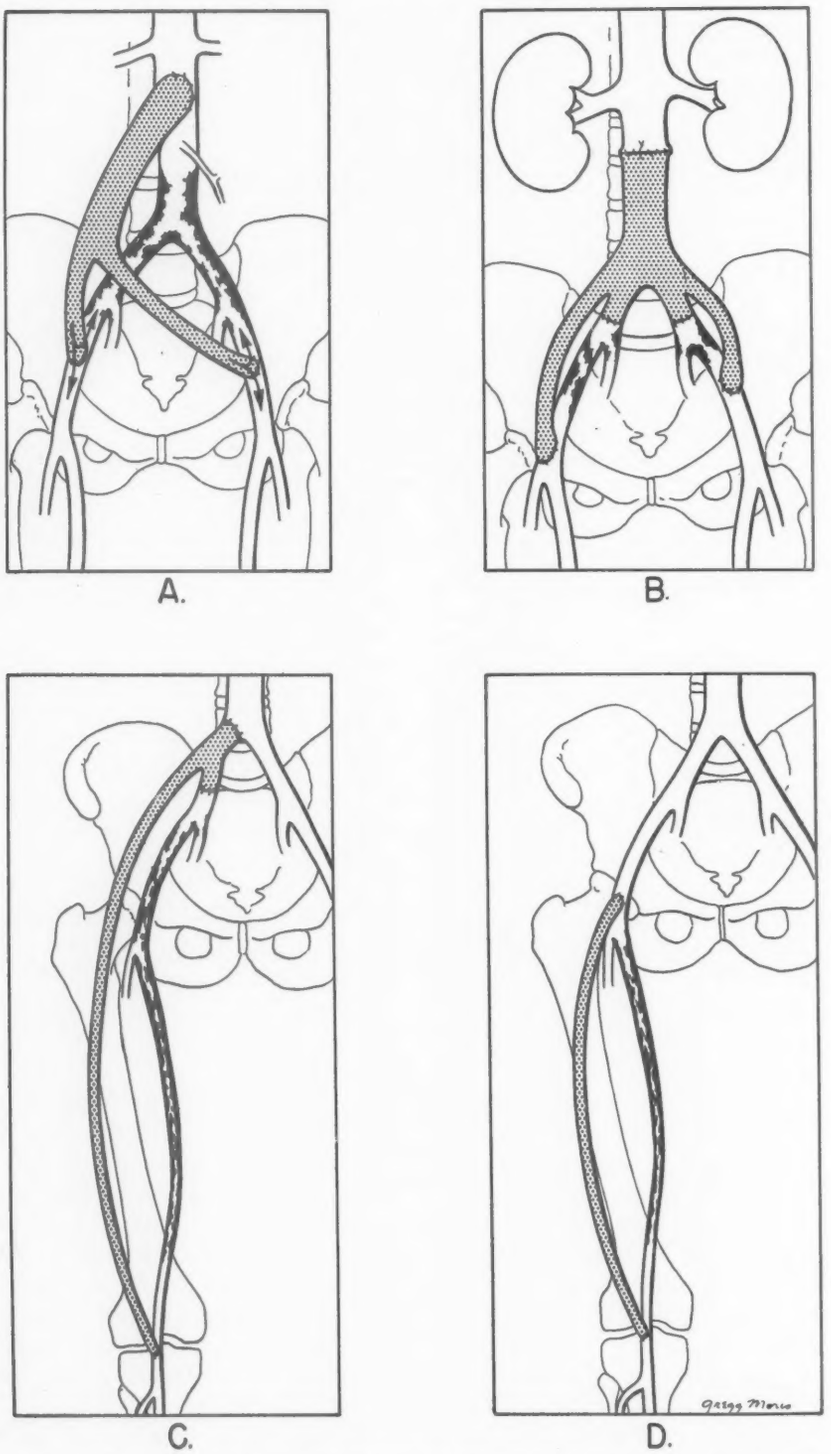


FIG. 1. Illustrated are situations where the bypass graft technic can be utilized. A, "piggy back" graft used to bypass a stenosed abdominal aorta and the common iliacs. B, resected aortic bifurcation with insertion of homograft; internal iliac of graft anastomosed to host common iliac. External iliac of graft anastomosed to left external iliac and right common femoral. C, bypass graft from aorta to popliteal with preservation of flow through internal and external iliac arteries. D, typical femoral-popliteal bypass graft.

on the first postoperative day and the other clot was cleaned out of the popliteal artery distal to the lower anastomosis. One limb required amputation. Four patients had the abdominal aorta resected below the renal arteries and an aortic bifurcation graft inserted. (Fig. 1B.)

TABLE II
ADDITIONAL SURGICAL PROCEDURES

Sympathectomy	8
Thromboendarterectomy	5
Thrombectomy	2
Amputation	1
Appendectomy	3
Bifurcation graft	4
Total	23

Every patient complained of intermittent claudication of varying intensity in the buttock, hip, thigh or calf, depending on the site of obstruction. It is possible to localize with a reasonable degree of accuracy the site of arterial obstruction by the distribution of claudication pain [5]. Eighteen of the twenty-three extremities were painful at rest, a symptom which usually indicates high grade obstruction with inadequate collateral circulation [14]. With few exceptions the extremities were cool and occasionally associated with numbness or tingling. In only eight was any change in color noted. Two had ischemic ulceration, one on the shin and the other on a toe. The latter was initiated by trauma and aggravated by infection. Five patients had retarded growth of hair or toenails.

After a complete clinical examination the patients were studied in the cardiovascular laboratory. The laboratory examination consisted of studies carried out by employing the vasograph.* This instrument consists of a console which contains three separate units: a segmental limb plethysmograph, a digital plethysmograph and three thermistor thermometers. The segmental limb plethysmograph was used for measuring the pulsations (volume changes with each pulse beat) of the large arteries of the arms and legs. These volume changes were recorded in terms of milliliters per pulse beat. This instrument is equipped with a standardizing device so that volume changes are recorded, and thus differs from the conventional oscillometer which does not record volume change and which gives results in arbitrary, unstandardized units. The digital plethysmograph was utilized to measure systolic blood pressure at various levels on the arms and legs. Also, the pulse waves and blood flow of the fingers and toes were recorded, which made it possible to study the circulation through the small terminal vessels. The thermistor thermometers were employed to study the circulation to the skin of the digits and to indicate when vasodilation was at its height. These studies were made before and after vasodilation produced by a posterior tibial nerve block.

* Electro-Medical Engineering Co., Burbank, Calif.

TABLE III
RESULTS

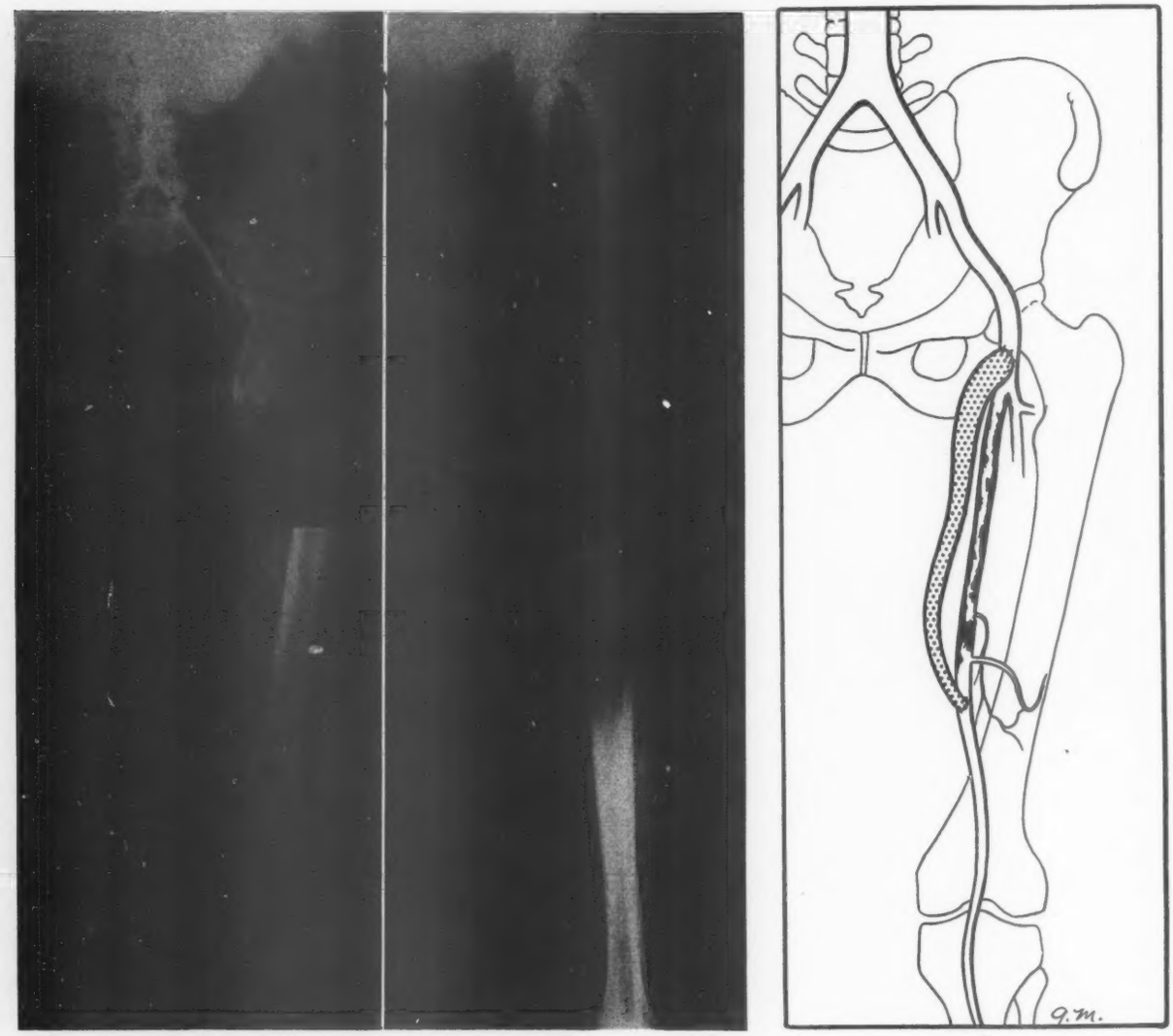
Classification	Initial	Late
Excellent	17	19
Improved	2	2
Poor	4	2

The results were classified as excellent, improved and poor. (Table III.) The criteria for each classification follow. The excellent category included cases in which there was (a) palpable pulsation over the graft and return of palpable pulses distal to the obstruction, (b) vasographic improvement, and (c) disappearance of intermittent claudication with production of an asymptomatic, functional limb, including healing of skin ulceration. Cases were recorded as improved if (a) palpable pulsations distal to the obstruction were absent but present over the graft, (b) the vasographic studies gave evidence of significant improvement, and (c) clinical improvement occurred. Results were interpreted as poor if there were (a) no pulsations over the graft or return of pulsations distal to the obstruction, (b) no significant improvement demonstrated with the vasograph, and (c) no clinical improvement.

Preoperative arteriograms were taken in all cases. Postoperative arteriograms were taken in only four cases because postoperative vasographic studies were considered safer and in many respects superior to angiograms.

TECHNIC

The principles and advantages of the end-to-side bypass graft have been described ade-



2A 2B 2C

FIG. 2. A, preoperative angiogram of obstructed left superficial femoral artery. B, postoperative angiogram of femoral-popliteal bypass. C, artist's conception of the bypass graft. Note the near right angle with which the collateral branches leave and enter the main arterial trunks.

quately [3,4,7,9–11,15]. With few exceptions the technics reported in these references were used in this series of patients. These differences will be indicated. The end-to-side anastomosis may be accomplished with the same ease as an end-to-end anastomosis. The proximal end of the obstruction dictates where the graft must be inserted. A patent popliteal artery distal to the obstruction insures an adequate arterial outflow and provides a reasonable chance for a successful outcome. One disadvantage was noted when the graft was carried across a flexion crease. (See case report.)

A striking clinical observation is the infrequent occurrence of obstruction in the arterial branches which come off at right angles, namely, the thoracic intercostals, the lumbars, the renals and the anterior tibial artery. Many collateral vessels leave and enter the large arterial trunks at almost 90 degrees. (Fig. 2.) In an attempt to duplicate nature, the end of the graft was inserted into the side of the host artery at an angle between 45 and 90 degrees. Technically it is easier to accomplish and maintain the anastomosis at the proper angle if the bypass graft is brought down the thigh in the subcutaneous layer rather than deep to the fascia. (Fig. 3.)

Since the obstruction of the lower abdominal aorta and the iliac arteries was incomplete in

two patients, it was possible to bypass rather than resect the bifurcation. The proximal end of the aortic bifurcation graft was anastomosed to the side of the host aorta. (Fig. 1A.) In one case the distal end-to-side anastomosis was at the external iliacs. In the other case the distal anastomosis was accomplished bilaterally end-to-side into the common femoral artery, below the inguinal ligament.

When the aortic bifurcation was found to be almost completely obstructed, it was resected and the graft inserted into the aorta with an end-to-end anastomosis. (Fig. 1B.) The external iliac branch of the graft was sutured end-to-side to the host's external iliac or common femoral, or superficial femoral or popliteal, depending on the location and distal extent of the obstruction. Instead of closing off the host's common iliac, this segment was thromboendarterectomized and sutured to the internal iliac branch of the graft. In this manner maximum flow through the internal and external iliacs and previously established collaterals was preserved and utilized.

When a femoral-popliteal bypass graft was inserted, the proximal anastomosis to the common femoral artery was end-to-side. (Fig. 1D.) This procedure insured an adequate inflow, provided an excellent angle for the anastomosis and was above the profunda branch. The fact that the anastomosis was above the profunda is important because of the high instance of impaired flow through the superficial femoral artery distal to the profunda branch. The upper end of the graft was not placed in the superficial femoral artery because of the poor inflow.

The distal anastomosis also was performed end-to-side. The lower end of the graft should be smaller in diameter than the proximal end. However, the distal end usually is an iliac artery and is therefore larger than the host's popliteal artery. A patent popliteal artery with good backflow is desirable if a successful result is to be obtained. However, nothing can replace meticulous surgical technic as a major factor in determining the ultimate success of a graft. In Figure 2 are shown pre- and postoperative angiograms together with a drawing of the bypass graft.

RESULTS

Of the twenty-three extremities treated with bypass technic using lyophilized homografts, the initial results were considered excellent in seventeen. Two patients were classified as improved. In four patients the initial results were poor. Two of the patients included in the poor group had immediate revision of their grafts and are now in the excellent classification. (Table III.)

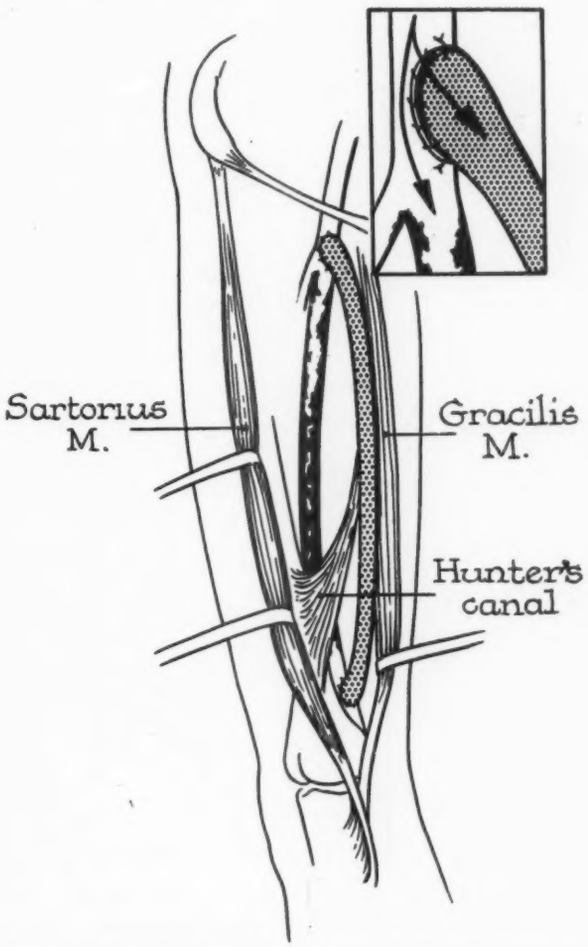


FIG. 3. Femoral-popliteal bypass graft in subcutaneous layer. *Insert*, note the angle (45 degrees or greater) of the anastomosis.

Vasographic examination of two of the four grafts with poor results suggested that arterial disease was present distal to the popliteal bifurcation prior to the surgery. One of these patients required amputation. Examination of the amputated limb substantiated the previous impression of marked arterial disease distal to the popliteal artery. In this case the bypass was unsuccessful in preventing or lowering the site of amputation.

Two of the patients in the poor group required revision of the grafts during the immediate postoperative period and were converted

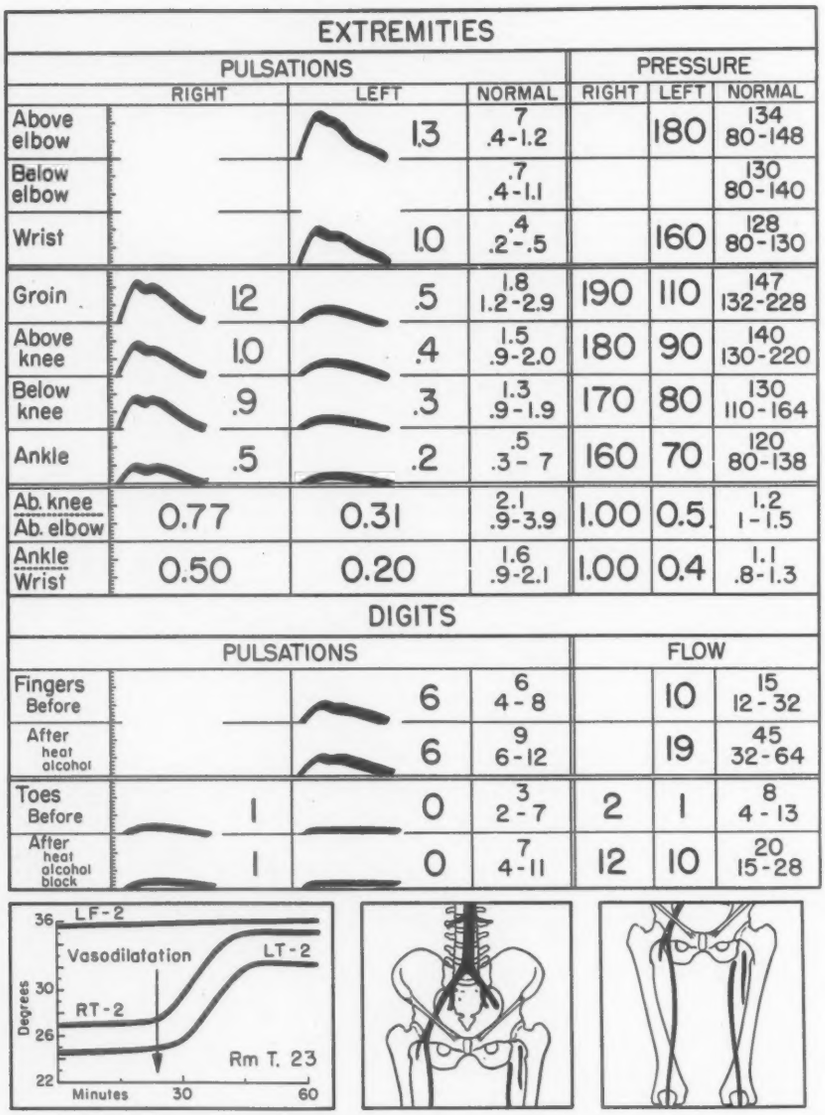


FIG. 4. Patient R. C. Preoperative vasographic study.

to the excellent group. The progress of one of these patients is described in detail:

R. C., a forty-eight year old man, complained of pain in the left hip and thigh after walking one block. Preoperative vasographic examination of the left leg (Fig. 4) showed low pulse waves at the groin, above the knee, below the knee and at the ankle. These waves were rounded and had no dicrotic notch. The pulsation indexes (height of pulsation in the leg divided by the height of pulsation in the arm) were abnormal and indicated arterial disease of both lower extremities. The left leg was more severely involved than the right.

An evaluation of the pulsations in the index finger and in the right and left second toes also revealed disease of the left side. The pulsations in the left toe were absent before and after the following vasodilating procedures: indirect body heat, the consumption of 2 ounces of alcohol and a posterior tibial nerve block.

The systolic blood pressures in the entire left leg were low as compared with those in the right. The blood flow through the left second toe was 1.0 cu. mm. per second before vasodilation and increased ten times after dilation. This increase, however, brought the flow only to about half of the normal average value. The blood flow through the right toe was greater than the left.

The skin temperatures of the left toe were lower than those on the right. However, the temperatures on the left increased 7°c. with the vasodilating procedure.

The angiogram demonstrated an obstructing

EXTREMITIES							
	PULSATIONS				PRESSURE		
	RIGHT	LEFT		NORMAL	RIGHT	LEFT	NORMAL
Above elbow		1.4		.7 .4-1.2		180	134 80-148
Below elbow				.7 4-1.1			130 80-140
Wrist		1.0		.4 .2-.5		162	128 80-130
Groin	1.3	1.4		1.8 1.2-2.9	190	194	147 132-228
Above knee	1.1	1.3		1.5 .9-2.0	182	185	140 130-220
Below knee	1.0	1.2		1.3 .9-1.9	171	175	130 110-164
Ankle	.6	.8		.5 .3-.7	160	160	120 80-138
Ab. knee / Ab. elbow	0.79	0.93		2.1 .9-3.9	1.01	1.0	1.2 1-1.5
Ankle / Wrist	0.60	0.80		1.6 .9-2.1	0.99	1.0	1.1 .8-1.3
DIGITS							
	PULSATIONS				FLOW		
Fingers Before		5		6 4-8		15	15 12-32
After heat alcohol		5		9 6-12		15	45 32-64
Toes Before	1	6		3 2-7	1	5	8 4-13
After heat alcohol block	1	6		7 4-11	1	18	20 15-28

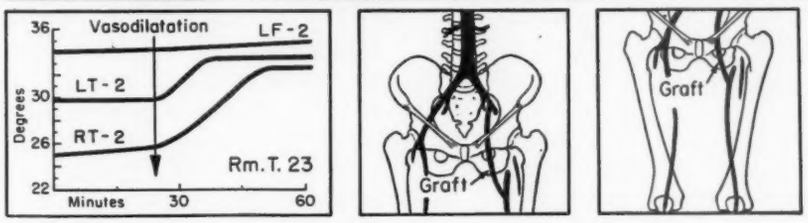


FIG. 5. Patient R. C. Postoperative vasographic study.

lesion in the left external iliac, common femoral and a portion of the superficial femoral and the profunda arteries, with refilling below the obstruction.

A bypass graft was inserted from the patent left external iliac artery to the superficial femoral artery, passing under the inguinal ligament. This graft was an immediate failure. On the first postoperative day the patient required revision of the graft. Excellent distal flow was re-established by extensive thrombectomy of the graft using a combination of methods, namely, direct removal, retrograde washing and catheter aspiration. The graft then remained patent for six months. Three months after the revision vasographic examination (Fig. 5) showed a marked increase in the height of the pulsations of the left leg, and an increase in the systolic blood pressures, pulsations and blood flow on the left. The blood pressure and the pulsation indexes were improved. Six months after the surgery, thrombosis of the homograft occurred and the patient's vasographic examination was similar to the one made prior to the first graft. This finding suggests that blood was coming through the bypassed major channels and collaterals, although the graft was thrombosed. The patient had been sitting on a pier railing with the thigh sharply flexed on the trunk for about two hours. When he stepped down from this position the left leg and foot were "asleep" and the previously palpable pulsations had disappeared. This situation represents the only complication in this series resulting from a graft being carried across a flexion crease. The patient was returned to surgery and the graft replaced. The second graft has re-

mained patent in a functioning, asymptomatic limb for the past five months. It should be noted that this patient was on dicumarol with a prothrombin level of 18 per cent at the time of the thrombosis of the graft.

Another one of the four patients with poor results had a graft inserted from the right common femoral to the popliteal artery. This graft thrombosed on the third postoperative day at the time of ambulation. The graft was removed that day and a new one inserted which is now open. The leg is asymptomatic and functional, and the patient is included in the excellent group. The thrombosis started at the distal anastomosis, low in the popliteal artery, and was observed as it propagated up the thigh. It may be significant to note that the thrombosis occurred where the graft entered the popliteal artery at an acute angle.

The fourth patient in this group died on the third postoperative day from renal failure and toxemia due to a gangrenous leg. This patient was obviously a poor selection for surgical therapy because the preoperative vasographic studies recorded very low pulsations, blood flow and skin temperatures through the toes with no element of vasoconstriction. However, this was a series of unselected patients and no one was refused the possible benefits of surgical therapy.

COMMENTS

Survival of the ischemic limb is not the only criterion for a successful graft [*14*]. Every effort must be made to produce a functioning, asymptomatic extremity. The bypass homograft is a satisfactory method for re-establishing the circulation to the limb distal to an arteriosclerotic obstruction of the common iliac, external iliac, internal iliac, common femoral, profunda femoris and the superficial femoral arteries. The bypass technic also preserves and utilizes the previously established collateral circulation. It is especially important to preserve circulation in the internal iliac artery; otherwise intermittent buttock and hip claudication will not be relieved. The bypass procedure proved to be less satisfactory for the patient with obstruction distal to the popliteal artery and its bifurcation. The poor results obtained in the obstructions of the popliteal artery and vessels below the knee were due most likely to the inadequate arterial run-off. In this situation the graft is almost certain to thrombose in the early postoperative period.

The essentials for a successful bypass graft are: (1) a suitable graft, (2) establishment of sufficient inflow, (3) the presence of adequate arterial run-off, (4) the maintenance of an adequate systolic blood pressure during and after surgery, and (*5*) a technically well done anastomosis. Early failure of a graft is usually caused by the absence of one or more of these essentials. Two additional factors influence late failures: (1) progression of the original disease in the host artery, and (2) structural changes that may exist or develop in the graft [*15*]. Unless precautions are taken, a diseased artery may be used inadvertently as a graft. All grafts in the artery bank should be x-rayed so that those containing calcium may be identified and discarded. (Fig. 6.)

The administration of anticoagulants in the postoperative period does not protect the patient against thrombosis. Thrombosis of the graft developed in the patient who received dicumarol even though his prothrombin time was within the therapeutic range. The dicumarol was administered to this patient because of a coronary occlusion with myocardial infarction which the patient had had several months before surgery.

Many vascular surgeons believe that angiography offers the greatest assistance in determining operability and in following the patient's postoperative progress [*15,17,19*]. Long film technics and serialized exposures are necessary to give an acceptable picture of a diseased arterial tree [*13,17,18*]. Anything less is a compromise. Every patient reported in this series had a preoperative angiogram. Only four had a postoperative angiogram but eleven had postoperative vasographic studies.

Vasographic studies provide more accurate information than the angiogram regarding the run-off and the functioning collateral circulation [*16*]. The vasographic examination makes it possible to improve the selection of patients for bypass grafts. If the circulation through the toes is studied before and after posterior tibial nerve block and measurements of the volume of pulsations, relative blood flow and skin temperatures of the toe are made, it is possible to determine rather accurately whether circulation through the calf and foot is adequate for survival and function. When the circulation is improved after posterior tibial

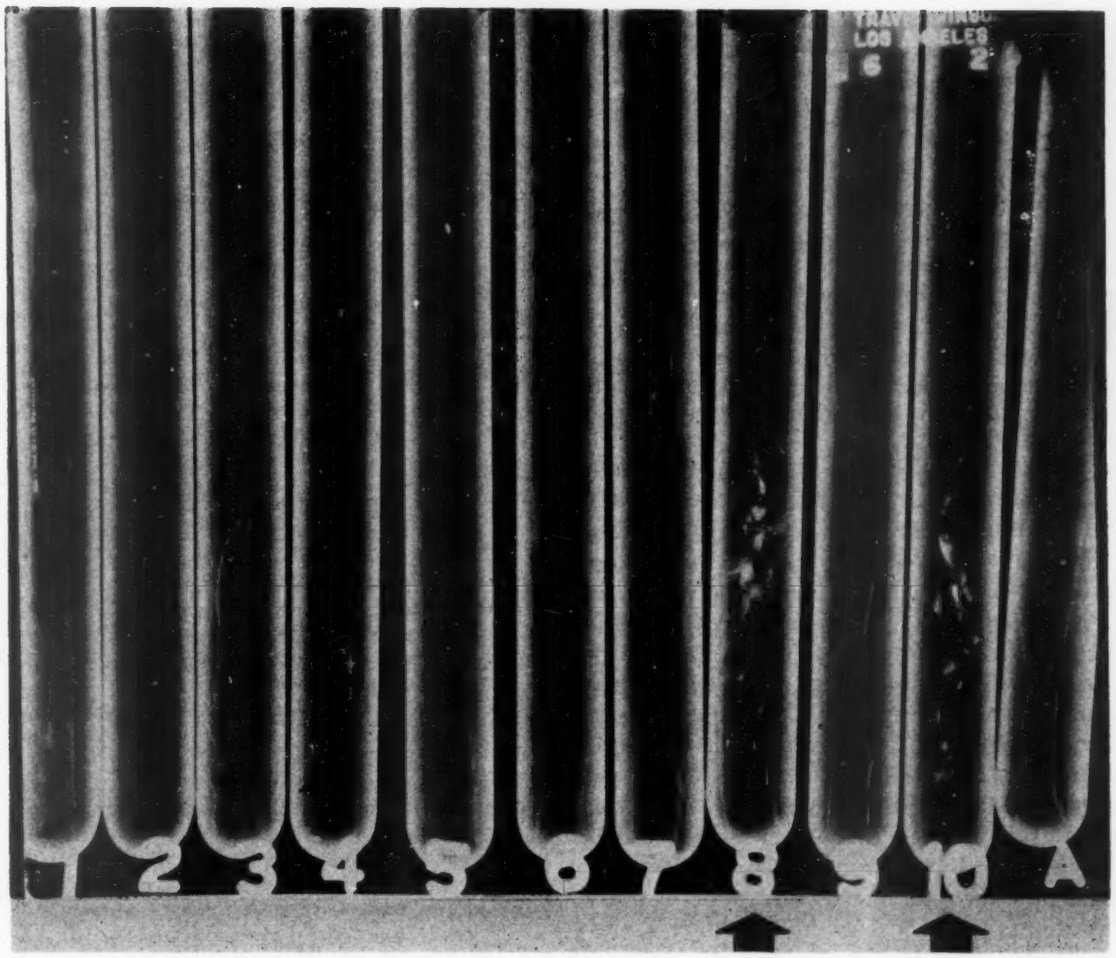


FIG. 6. Roentgenogram of homografts in the Los Angeles County Heart Association Artery Bank. Note calcium deposits in grafts 8 and 10.

nerve block, it is logical to assume that the smaller vessels are patent and a bypass graft even to the arteries below the knee is likely to be successful. Vasographic studies also make it possible to determine the amount of functional vasoconstriction which is present in the distal parts of the extremities. In this study, when high grade functional vasoconstriction was present, lumbar sympathectomy was performed. There was no mortality or morbidity for the vasographic study.

The magnitude of the distal flow may be ascertained by measuring the toe pulse, blood flow and skin temperatures before and after a posterior tibial nerve block. For example, organic arterial disease without a functional vasoconstrictive component is probably present in a patient whose toe shows low pulse waves of only 2 or 3 cu. mm./second, and a low skin temperature of 26° or 27°C., and who after blocking shows no significant change in any of these measurements. In such a patient it is likely that a bypass graft below the popliteal artery will be unsuccessful because of the poor flow through the vessels distal to the graft. On the other hand, if the patient's toe shows low pulsations, blood flow and skin temperatures before block, and after the block shows high pulse waves of 7 cu. mm., a high blood flow of 20 cu. mm./second and a skin temperature of 36°C., it may be concluded that a strong functional vasoconstrictive element is present with little organic disease of the arteries and that distal flow is good. In this situation a sympathectomy and bypass graft would be likely to succeed.

Vasographic examinations are valuable in following patients during the postoperative period. Examinations may be done at frequent intervals without harm or discomfort to the

patient, and objective measurements may be obtained. It should be pointed out that the height of the pulsations from the limb obtained with the segmental plethysmograph is a standardized reproducible record of volume change per pulse beat which differs from the measurement made with the conventional oscillometer which is not reproducible because it is not properly standardized. Because of the standardization, differences in the tightness of the cuff and such factors as the age of the tubing and rubber parts of the instrument do not introduce errors into the readings.

SUMMARY

Arteriosclerotic arterial insufficiency of the peripheral blood vessels is often due to localized obstruction with a patent arterial system above and below the obstruction. The results of twenty-three bypass grafts in fifteen patients are reported. The clinical and laboratory methods of diagnosis, management, surgical technic and follow-up are described.

The principles and advantages of the end-to-side bypass graft are presented. Of the twenty-three extremities treated with the bypass graft technic using the lyophilized homograft, the initial results were excellent in seventeen, improved in two and poor in four. Two patients in the last group had their grafts revised or replaced and are now in the excellent group. Our results as of now are: nineteen excellent, two improved and two poor. No patient's blood flow was impaired by the surgical procedures, although we did fail to prevent an amputation. There was one death.

A longer follow-up is necessary before the ultimate fate of the grafts and the final outcome of these patients will be known. However, this study indicates that the lyophilized homograft is a suitable material for bypassing arterial obstructions. In addition, the bypass graft technic is an acceptable method for the treatment of segmental occlusion of the common iliacs, internal iliac, external iliac, common femoral and superficial femoral arteries. This technic may be employed for obstruction in the popliteal artery provided the distal arteries are patent and can maintain a good blood flow to the limb after the graft is in place.

The vasographic examination, which is an integrated laboratory technic for examining the peripheral circulation, proved of value in the selection of patients for the bypass graft and for lumbar sympathectomy. Vasograms were of value also in following the progress of patients at frequent intervals without mortality, morbidity or discomfort to the patient. The vasographic examination made postoperative angiograms unnecessary.

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DISCUSSION

EDWIN J. WYLIE (San Francisco, Calif.): This challenging opportunity to discuss the exciting work of Dr. Payne and his colleagues also represents an opportunity to compare varying approaches to a common problem. It is necessary, however, that there be common criteria in order to make such comparisons valid. The many variations in the degree of severity of proximal and distal occlusive disease in segmental or arteriosclerotic thrombosis and the varying effect of sympathetic interruption (whether by lumbar sympathectomy or merely by the division of sympathetic fibers coincident to local artery dissection) may influence peripheral symptoms or the laboratory assessment of peripheral flow. For these reasons, my remarks are limited to a discussion of the functioning of the arterial substitute alone.

Dr. Payne has indicated that early thrombosis appeared in three grafts (13 per cent), although in six (26 per cent) palpable pulsations were absent after operation. In our experience the absence of postoperative pulses in the femoral artery following aorta-iliac or aorta-femoral bypasses, and in the popliteal artery following femoral bypasses has been due in each instance to graft thrombosis. A thigh pulse following a successful femoral bypass is extraordinarily easy to feel if the graft has been placed in a more superficial position as Dr. Payne has shown.

Some of our patients with graft thrombosis nevertheless have shown clinical improvement possibly for the reasons indicated heretofore. Even with the adoption of this more rigid criterion, the resulting 26 per cent thrombosis rate compares with that recorded in other published series. We have also concluded that patency of the peripheral outflow tract is the most important factor in preventing graft failure. Dr. Payne and I disagree to some extent concerning the method of graft insertion. Inasmuch as turbulence of blood flow appears to be a factor in producing thrombosis, we have favored a graft insertion using as oblique an angle as possible. We actually anastomose the graft with some degree of tension in order to align the peripheral artery with the long axis of the graft.

Thirty-one bypass arterial grafts have been implanted in patients at the University of California Hospital during the past fourteen months. Sixteen grafts were prepared by beta-propyl-lactone sterilization and buffered electrolyte solution preservation, and fifteen by 70 per cent ethyl alcohol sterilization and preservation. Eighteen were aorta-to-femoral grafts for the surgical treatment of advanced occlusive disease involving the terminal aorta and the full extent of the common and external iliac arteries. Thirteen were common femoral-to-popliteal grafts. Immediate thrombosis appeared in seven patients of this series, three of which, however, involved only one arm of an aorta-to-bilateral femoral graft. In the last three cases we reoperated as soon as the diagnosis of thrombosis was made, and patency was regained in all three. There were two cases of late thrombosis, at four and eight months, respectively. We believe these to have been due to technical errors which caused constriction at the site of anastomosis.

The over-all thrombosis rate of 23 per cent in our experience with bypass grafts has been compared to the thrombosis rate in our previous thromboendarterectomies. In a series of 100 consecutive thromboendarterectomies for treatment of thrombosis or stenosis involving the aorta and common iliac arteries, immediate thrombosis developed in seven, of which four involved only one iliac limb. In none of the seven was ischemia increased as a result of operation. In the last fifty operations thrombosis has occurred only once. There have been no late thromboses in this group during the follow-up period ranging from three months to six years.

A more comparable series, however, is an additional group of thirty-one patients who underwent thromboendarterectomies for the advanced type of lesions which Dr. Payne has been discussing and for which we have also been performing bypass grafts. These lesions include stenotic or thrombotic involvement of common and external iliac arteries, external iliac and common femoral arteries, or long lengths of the superficial femoral arteries. Immediate thrombosis occurred in eight patients (26 per cent) of this group, and late thrombosis in four, all within the second year after surgery.

It is of interest to note that we have improved our salvage rate only slightly by adopting the bypass method in this more advanced type of disease. The relative ease of bypass operations in patients with extensive occlusion and the lesser likelihood of disturbing existing collateral pathways would seem nevertheless to make this the preferable procedure. For the simpler lesions of aorta–common iliac thrombosis, we clearly believe thromboendarterectomy to be the better procedure.

ROBERT A. WISE (Portland, Ore.): May I congratulate Dr. Payne on his brilliant paper and Dr. Wylie on his important discussion. I would like

to call attention to the fact that occasionally the bypass procedure is of value in acute traumatic arterial injury.

We had such a situation three years ago, when the superficial femoral artery had been divided and ligated exactly at the division of the common femoral into the profunda and superficial femoral arteries. Distally the superficial femoral artery had been ligated after a loss of approximately 6 cm. of the artery.

The patient was seen one hour after the accident. It became evident that in order to establish continuity of the superficial femoral artery by a graft it would be necessary to divide the common femoral and sacrifice the profunda femoris should an end-to-end suture be employed. If the graft thrombosed, we would have complete loss of both the profunda and superficial femoral artery circulation. Therefore we decided to use a bypass graft between the common femoral artery proximal to the profunda, and the superficial femoral artery.

Films* showed the divided superficial femoral artery at the bifurcation of the common femoral artery. The profunda femoris was intact. The graft extended from the common femoral artery, where it was anastomosed end-to-side, to the distal end of the divided superficial femoral artery.

On a femoral arteriogram taken several months following operation the graft which had restored continuity to the superficial femoral artery could be noted. It was also noted that the profunda femoris artery circulation was preserved.

This case illustrates the use of a bypass graft in acute traumatic division of an artery at a major branch, thereby preserving the circulation which might be sacrificed by an end-to-end technic.

The use of a bypass graft in arteriosclerotic occlusion of the external iliac artery is illustrated by another patient. This woman had severe claudication in her left lower extremity, being able to walk only 50 feet without pain.

An aortogram showed complete occlusion of the left external iliac artery. The arteries on the right showed minimal changes.

At operation a bypass arterial homograft was placed between the left common iliac artery and the left common femoral artery using the end-to-side technic.

A postoperative arteriogram was taken two years following surgery and showed the patent arterial homograft extending from the left common iliac artery to the left common femoral artery.

The patient states that she can walk approximately 1 mile without signs of claudication. Through the use of a bypass graft collateral vessels were uninjured and adequate arterial blood was provided to the left lower extremity.

* Dr. Wise illustrated his discussion with slides.

J. Karl Poppe (Portland, Ore.): I would like to ask Dr. Payne how he obtains long homografts in sufficient numbers.

I have had considerable difficulty obtaining homografts of sufficient length to reach the mid-thigh region from the mid-abdominal aorta. It is only by a great deal of persuasion and long delays that these are obtained, while the patient postpones his operation.

It seems particularly desirable under such circumstances to use the graft before dry freezing, so that any few centimeters of femoral artery which are not used can be preserved for the next patient.

J. Howard Payne (closing): I wish to thank Dr. Wylie for his kind and informative discussion, and the other participants for their interest in this presentation.

In regard to the question of obtaining grafts, I am sure it is a difficult problem in any city. We obtain most of our material from the Los Angeles County Hospital and the Hospital of the Good Samaritan. The residents in surgery have the responsibility of obtaining the specimens with the cooperation of pathology residents. If the surgical resident would go to the autopsy room to assist the pathologist or take the arteries himself, an adequate supply could be assured. We have two surgical residents on call for the autopsy room. The secretary advises them when a suitable postmortem examination is to be performed. The surgical resident either takes the arteries or assists the pathologist. It is true that in each hospital we have a very cooperative pathology department. This explains the reason that our bank is well stocked.

With reference to the angle of anastomosis, it is interesting to note that in two cities as close as San Francisco and Los Angeles we are approaching the problem from different angles. We have attempted to duplicate the angle of the anastomosis as it occurs in nature. It is evident that the right angle branches of the aorta, the renals, the lumbars, the thoracics and an outstanding example, the anterior tibial artery, are rarely the site of obstruction. The majority of the arterial obstructions begin at the branches with acute angle origin; the internal iliac artery is one of the most notable. We are fully aware that these clinical observations may be fallacious. The School of Engineering has been consulted in an effort to clarify the problem of the proper angle of anastomosis. Time and accurate laboratory study will be necessary before final conclusions can be made. To date our results, using the methods and materials described, have been most gratifying.

Primary Cardiac Tumours

SURGICAL TREATMENT

ROSS ROBERTSON, M.D., *Vancouver, British Columbia, Canada*

CARDIAC tumours became of increasing interest to cardiologists following an excellent study and review of the literature by Lisa, et al. [*16*] in 1941. These workers outlined the outstanding features of cardiac tumours which should lead to the diagnosis.

Successful surgical excision of a cardiac tumour was first reported by Beck [*2*] in 1942. He discussed heart tumours in general and suggested that myxomas of the left atrium could be removed. Stimulated by his report, numerous attempts were made to excise cardiac tumours, with a few successes [*5,7,17,20*].

Following are case reports on five patients with cardiac tumours treated surgically.

It is convenient for the surgeon to classify cardiac tumours as rising from the pericardium, myocardium or endocardium, since the diagnosis and treatment depend largely on their location.

PERICARDIAL TUMOURS

Pericardial tumours such as intrapericardial teratoma, lipoma, mesotheliosarcoma, hemangioma and lymphangioma may cause symptoms of constrictive pericarditis, obstruction of the vena cava or pericarditis with effusion. Cardiac x-ray films may show a rounded shadow merging with the heart or a greatly enlarged globular heart suggesting pericardial effusion. Aspiration of fluid, especially bloody effusion, assists greatly in the diagnosis. Maurer [*17*] has suggested that a pneumopericardium would have assisted in the preoperative diagnosis in his case. In 1952 he successfully removed a lipoma from within the pericardial sac attached to the anterior surface of the left ventricle.

CASE 1. (*Teratoma of pericardium.*) T. F., age two years, was admitted to the hospital in May, 1952, because of persistent cough and vomiting for ten months. X-ray study of the chest showed a rounded tumour along the right heart border. (Fig. 1.) The preoperative diagnosis was mediastinal dermoid cyst.

At operation in June, 1952, by Dr. R. H. Gourlay, a dumbbell-shaped tumour the size of two small

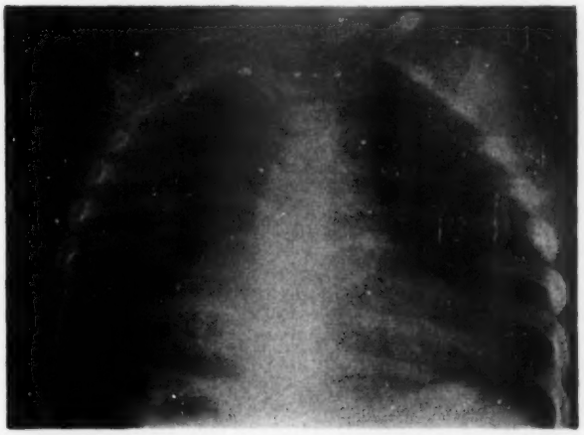

FIG. 1. Case 1. Teratoma of pericardium. Radiograph showing rounded tumour along the right heart border.

Japanese oranges was found lying between the greatly enlarged right atrium and the right ventricle, beneath the visceral pericardium. The tumour was attached to the right ventricle by a base measuring 2 inches in diameter. The right coronary artery was found embedded in the mass on three sides. The tumour appeared to obtain its blood supply from two large vessels springing from the right coronary artery at its origin. At this stage in the dissection cardiac arrest occurred but the beat was restored after five minutes of cardiac massage. After further dissection the tumour was removed but there was considerable bleeding from the branch of the right coronary artery entering the tumour. Cardiac arrest again occurred but the heartbeat was again restored rather quickly. Further bleeding from the right coronary artery was encountered and controlled by ligature with some difficulty. Cardiac arrest again occurred and the heartbeat could not be restored. It was thought

FIG. 2. Case I. Teratoma of pericardium. Low power view.

that the right coronary artery may have been occluded by the ligature.

The pathologic specimen was a dumbell-shaped mass measuring 8 by 4 cm. A small amount of heart muscle was attached to one surface. The other was covered by thickened pericardium. Microscopic sections showed a definite teratomatous type of growth characterized by gland formations, islands of cartilage, hair follicles, etc. (Fig. 2.)

CASE II. (*Cylindroma(?) of pericardium.*) V. S., a thirty-four year old woman, was first seen in October, 1949, complaining of increasing shortness of breath for three years and swelling of the ankles and abdomen. On examination the jugular veins were prominent. Venous pressure was 38 cm. The apex beat was not palpable. Heart sounds were distant and tic tac in quality. No murmurs were heard. There were moist rales at the base of both lungs. The liver was greatly enlarged. Cardiac x-ray films showed the heart shadow greatly enlarged, apparently outlining the pericardium. (Fig. 3.) Preoperative diagnosis was pericarditis with effusion.

At operation through the fifth left interspace

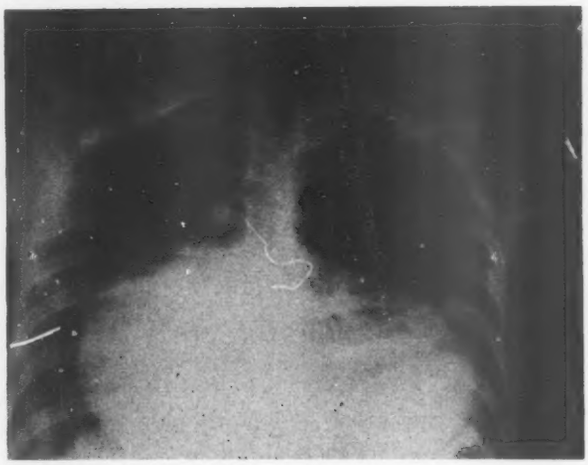

FIG. 3. Case II. Cylindroma (?) of pericardium. Radiograph showing huge globular heart.

anteriorly the pericardial sac was found to be huge and tense, and on opening it several hundred cubic centimeters of bloody fluid exuded. The heart was small. There were several tumour nodules, the largest measuring 2 cm. in diameter, scattered over the visceral and parietal pericardium. They were pale pink and located chiefly in the neighborhood of the pulmonary valve. A large window was left in the pericardium for drainage. The patient made a good recovery from operation. A section of the tumour was examined by several pathologists of the Tumour Registry, who gave a variety of diagnoses including cylindroma, metastatic adenocarcinoma and carcinoma. The patient remained well for five years following operation, when she finally died of local recurrence of the tumour. Autopsy was not performed.

CASE III. (*Mesothelial sarcoma of pericardium.*) A. N., a forty-seven year old woman, was first seen in April, 1956, complaining of retrosternal discomfort and dyspnoea since January. Before this she had felt well and had always been active in badminton and hill climbing. On examination the patient was in acute respiratory distress, being comfortable only in the knee-chest position. Her face was oedematous. The neck veins were full. The blood pressure was 100/80. A rub was heard over the pericardium. Heart sounds were distant. The liver was enlarged. Cardiac x-ray films showed a huge heart shadow apparently outlining the pericardium and occupying the lower part of the chest from side to side. (Fig. 4.) Pericardial paracentesis obtained 2,200 cc. of wine-coloured fluid. Four days later an additional 1,500 cc. of bloody fluid were aspirated.

On May 12th a left thoracotomy was performed. The pericardial sac was opened and drained of 2,000 cc. of bloody fluid. A white, fleshy mass of tissue surrounded and infiltrated the great vessels within the pericardium. It was not possible to remove the

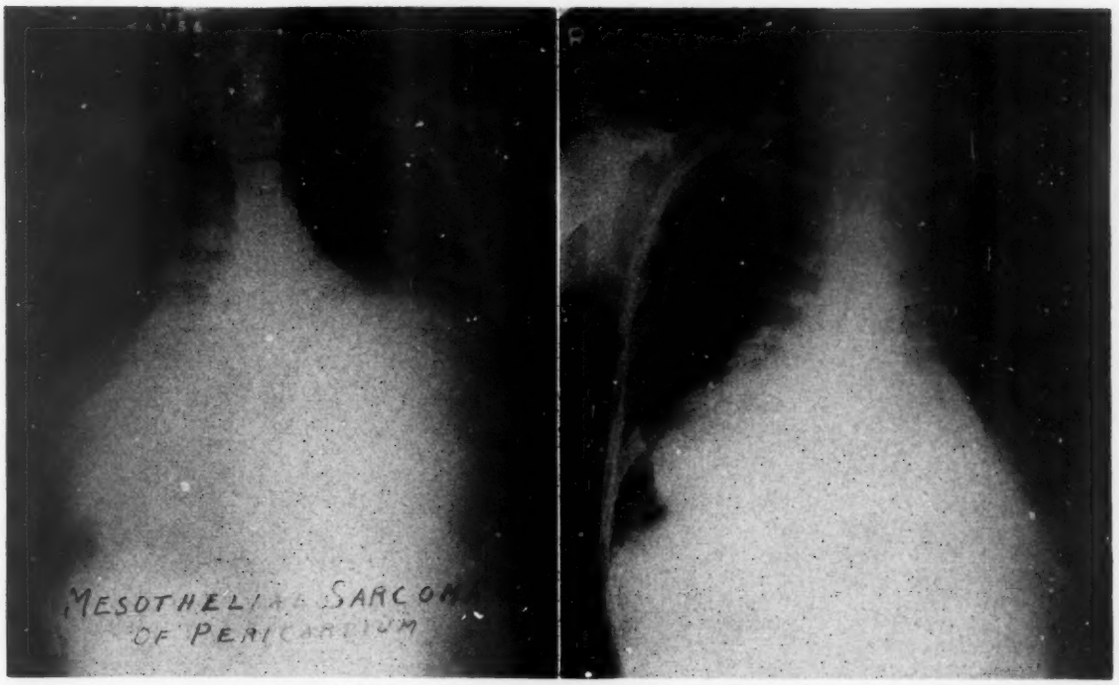

FIG. 4. Case III. Mesotheliosarcoma of pericardium. Pre- and postoperative radiographs.

tumour. A large window was made in the pericardium for drainage. Following operation the patient's condition deteriorated rapidly and she died on June 9th.

At autopsy the heart was grossly enlarged. There was widespread involvement of the pericardium by a malignant process. The tumour mass infiltrated the wall of the right atrium, protruding into the atrial cavity. Diligent search of the remainder of the body failed to reveal a primary site of the tumour. Microscopic sections showed sheets of undifferentiated pleomorphic cells containing scattered areas of necrosis, some of which were arranged in small groups which appeared to lie in preformed spaces, such as in lymphatic and blood capillaries The lesion was thought to be a mesotheliosarcoma of pericardial origin.

CASE IV. (*Lymphangioma of pericardium.*) R. G., age three years, was first seen in April, 1949. His parents stated that when the child was one month old he had had difficulty with his feedings and a diagnosis of congenital heart disease had been made. However, he was fairly well until March, 1949, when he became acutely ill with fever to 104°F., and signs developed of pleural effusion in the left chest. Pericardial aspiration recovered a small amount of thick, chocolate-like fluid, sterile on culture. X-ray films showed a tremendously enlarged heart with a lobular appearance in either border, suggesting an overlying thymus. The pericardial sac was again aspirated but no more fluid could be obtained. The diagnosis at that time

FIG. 5. Case IV. Lymphangioma of pericardium. Posteroanterior and lateral radiographs showing tumour with calcified margins, anterior to heart.

was myocarditis of unknown origin with pericardial effusion.

The child was followed up periodically and continued to be in fair health until 1956, when he complained of dyspnoea on exertion. He had no attacks of cyanosis. On examination the heart sounds were normal. There were no murmurs and rhythm was regular. Cardiac X-ray films showed a rounded area of increased density with calcification in the anterior mediastinum. (Fig. 5.) Tomograms and angiocardiogram revealed this to be an oval mass pressing on the superior vena cava, deeply indenting the right atrium and pressing upon the pulmonary artery and aorta which it displaced

FIG. 6. Case IV. Lymphangioma of pericardium. Appearance of resected tumour.

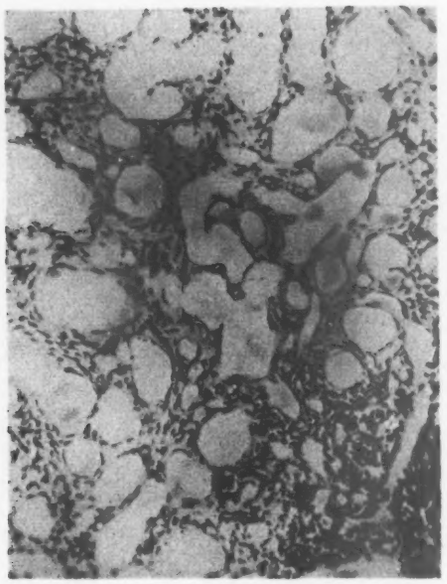

FIG. 7. Case IV. Lymphangioma of pericardium. Low power photomicrograph of resected tumour.

posteriorly and narrowed. The mass was presumed to be intrapericardial.

At operation on August 20, 1956, an incision was made across the sternum into the third right and left interspaces. The internal mammary arteries were doubly transfixed and ligated and divided. The pericardial sac was opened by a large H-shaped incision. Adhesions obliterated the pericardial space. A hard mass the size of a small orange was found attached to the pericardial sac, the right atrium, right ventricle, superior vena cava and aorta. It was freed with considerable difficulty from these structures. During the dissection the right coronary artery was encountered and a small artery appeared to branch from it into the cyst. This was ligated and divided and the right coronary artery protected. During dissection in this area the electrocardiograms showed changes of right coronary insufficiency. This disappeared when the dissection was concluded. In order to free the tumour from the aorta it was necessary to carry the dissection into the media of the aorta. Removal of the tumour left a depression in the right atrium and ventricle and between the superior vena cava and aorta, representing the bed of the cyst. Following its removal the anesthetist reported a rise in blood pressure to 160. The wound was closed in the usual way.

The patient made a good recovery from operation and when last seen in November, 1956, he was in good health.

The pathologic specimen (Fig. 6) showed a brownish tumour mass measuring 9 by 6 by 5 cm. The cut surface was brownish in colour and soft in consistency. It contained a few cystic spaces. There were some calcified areas in the capsule. Microscopic sections showed dilated lymphatic channels embedded in a loose connective tissue stroma in which lymphoid aggregates and reticulum cells were prominent. In the central areas there were large deposits of amorphous, hyaline granular material from old hemorrhages. The diagnosis was cystic lymphangioma. (Fig. 7.)

Comments. It seems likely that the original pericardial aspiration tapped an old hemorrhagic area within the tumour. It is of interest to compare this with Case 1, in which difficulty with the right coronary artery led to the child's death. The experience gained there assisted in the favourable outcome in this case. The unfortunate experience reported by Gebauer [*10*], in which uncontrolled bleeding from the aorta was encountered, led to increased care being taken in dissection in this area and contributed to the favourable result.

MYOCARDIAL TUMOURS

Tumours arising within the myocardium may account for arrhythmia, tachycardia or intractable cardiac failure without obvious cause. Most of the reported cases have been in infants or children. Bickford, Egan and Bryce [*3*] first made such a diagnosis during life in a two year old boy having recurrent bouts of tachycardia with failure. An angiocardiogram showed a large filling defect in the lateral wall of the left ventricle. The boy died suddenly at the age of

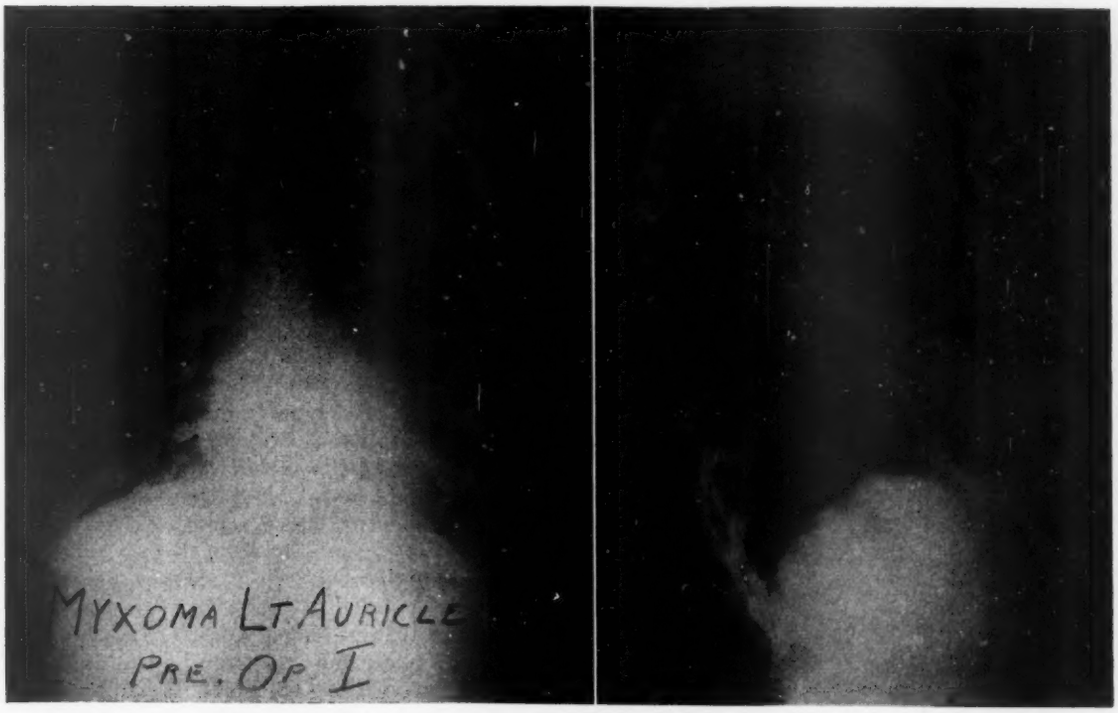

FIG. 8. Case v. Myxoma of left atrium. Posteroanterior and lateral radiographs before operation.

eight and a half years. The pathologic diagnosis was rhabdomyoma. Hurst and Copper [*13*] and Lisa et al. [*16*] report rhabdomyoma as being among the most common of heart tumours. We have not seen any rhabdomyomas at our hospital, possibly because they are usually associated with death in infancy.

ENDOCARDIAL TUMOURS

Myxomas form 50 per cent of the primary cardiac tumours and have become of considerable interest to the surgeon since the advent of mitral commissurotomy. The neoplastic nature of the myxoma remains suspect; some pathologists claiming that they are small organized thrombi. The comments of our pathologist which appear in Case v are of interest in this regard. Seventy-five per cent of myxomas occur in the left atrium, almost always attached to the fossa ovalis. They are usually polypoid and often pedunculated. There is little evidence that the heart has been damaged by previous disease. Prichard [*19*] stated in 1951 that no myxoma had been diagnosed during life, but since then Kirkeby and Leren [*14*] and Bahnson [*1*] made the diagnosis with the aid of angiocardiograms. In 1942 Beck [*2*] first suggested the possibility of surgical treatment for myxoma of the atrium, and recently a few myxomas have been removed successfully by Crafoord [*7*], Bigelow [*21*] and Scannel [*20*].

Myxoma of the left atrium should be considered in a patient with typical symptoms and signs of mitral stenosis who lacks any history of rheumatic fever or chorea. Usually the symptoms have appeared with unusual rapidity and fail to respond to medical treatment. They are often intermittent and may vary considerably with change in posture, apparently depending on the locale of the tumour, whether it is free in the atrium or obstructing the valve. Myxomas may occur in patients of any age. One has been described by Goldberg et al. [*11*] in a three and a half year old boy. Centeno [*6*] also reported a myxoma in a three and a half year old child with signs and symptoms of mitral stenosis. This child much preferred to lie in the prone position with the head low, differing from the usual patient with mitral stenosis. These patients may show signs of intermittent cardiac obstruction with sudden attacks of fainting, low blood pressure or even sudden death. In a patient with symptoms of tricuspid stenosis which is rarely a result of rheumatic endocarditis, myxoma should always be suspected.

Myxoma of the left atrium is frequently associated with embolic episodes, and it has been

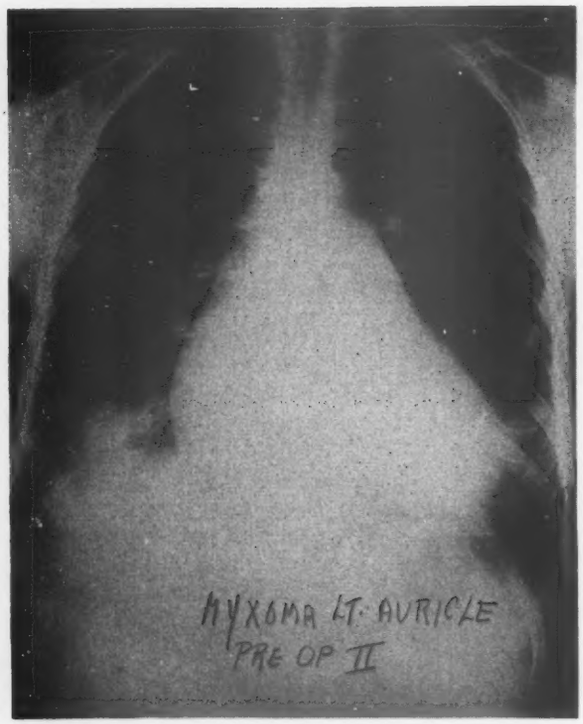

FIG. 9. Case v. Myxoma of left atrium. Radiograph before resection.

suggested that microscopic examination of such an embolus would prove the diagnosis. Cardiac rhythm has usually been found to be regular despite severe symptoms of mitral stenosis. The heart murmurs in myxoma of the atrium may vary considerably with the position of the patient along with the symptoms. Unlike mitral stenosis, the diastolic murmur of a myxoma which may be audible in the erect position may disappear with recumbency. There may also be a systolic murmur apparently from wedging of the tumour between the cusps, preventing complete closure.

CASE V. (*Myxoma of the left atrium.*) L. N., a thirty-eight year old woman, was first seen in February, 1955, complaining of increasing fatigue for one year and dyspnoea for three months. Cough and dyspnoea had rapidly become much more severe and there had been two or three slight episodes of hemoptysis. There was no history of rheumatic fever or chorea.

Examination with the patient in the recumbent position revealed a faint diastolic thrill over the apex and a harsh blowing mitral systolic murmur. A diastolic murmur could not be heard even in the left lateral position. The first heart sound was about normal in intensity. Cardiac rhythm was regular. The pulmonary second sound was accentuated. Cardiac x-ray films showed a slightly enlarged heart, the enlargement affecting the right ventricle and left atrium. The pulmonary vascular markings were accentuated. (Fig. 8.) A diagnosis of rheumatic heart disease with mitral stenosis was made,

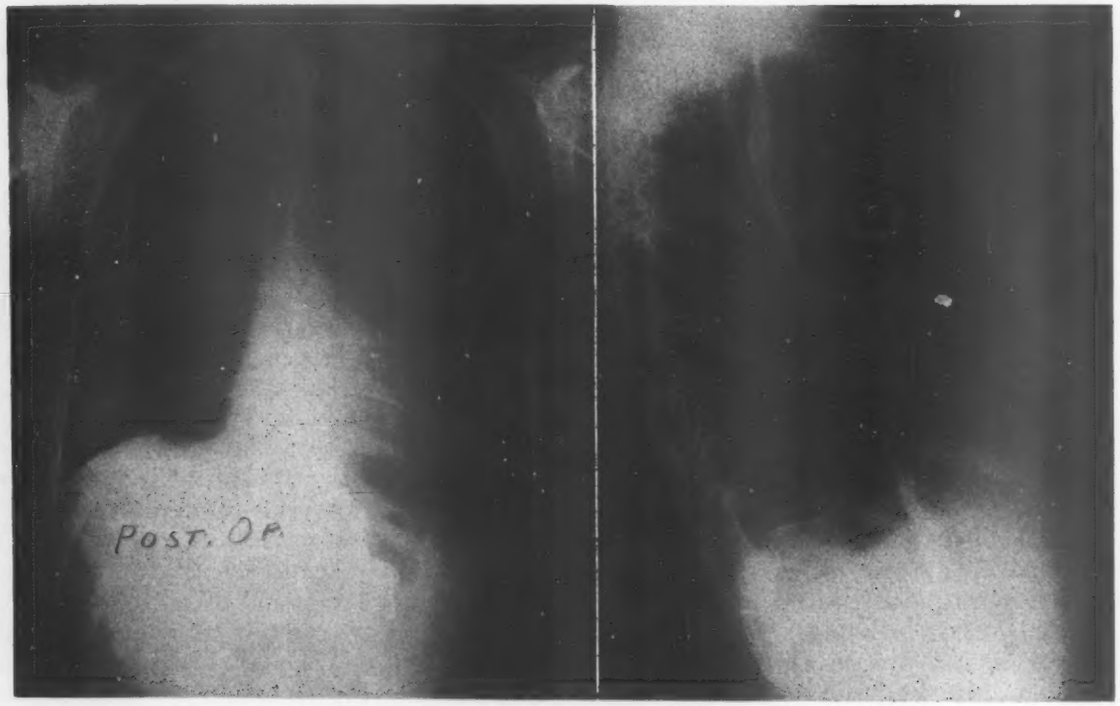

FIG. 10. Case v. Myxoma of left atrium. Radiographs after resection.

FIG. 11. Case v. Myxoma of left atrium. Resected tumour.

but the absence of the typical diastolic murmur cast some doubt on the diagnosis.

At operation for proposed mitral commissurotomy on July 7th the mitral valve was found to be normal. A rounded tumour mass about the size of a ping-pong ball was palpable in the left atrium. It was somewhat nodular and was attached by a pedicle to the auricular septum just above the aortic cusp of the mitral valve. It interfered with the closing of the aortic cusp, so that a moderate regurgitant jet of blood was palpable. It also seemed to obstruct the mitral valve orifice during diastole. A diagnosis of myxoma of the left atrium was made; and since preparations had not been made for dealing with this condition, the operation was discontinued and the wound closed.

The patient made a good recovery from the operation but her dyspnoea and hemoptysis persisted and she developed oedema of the ankles and an enlarged liver. X-ray study showed considerable increase in heart size, with increased pulmonary vascular markings and congestion at the right base. (Fig. 9.)

The patient's condition gradually deteriorated until June 21, 1956, when a second operation was performed for removal of the tumour under hypothermia down to 30°C. A trans-sternal incision was made into the fourth left and right interspaces. Adhesions from the previous operation presented some difficulties. The internal mammary arteries were doubly transfixed, ligated and divided. The pericardial sac was obliterated by fibrous adhesions, rendering dissection difficult. Two rows of stay sutures were inserted transversely in the left atrium and a rubber-covered clamp was applied to the atrium proximal to these sutures. A ¾ inch incision was then made between the suture lines through which the right index finger was inserted into the right atrium and the tumour mass palpated much as at the previous operation. It could be felt flapping up and down, in and out of the valve, and had a pedicle about the size of the index finger. The finger was withdrawn, the clamp reapplied and

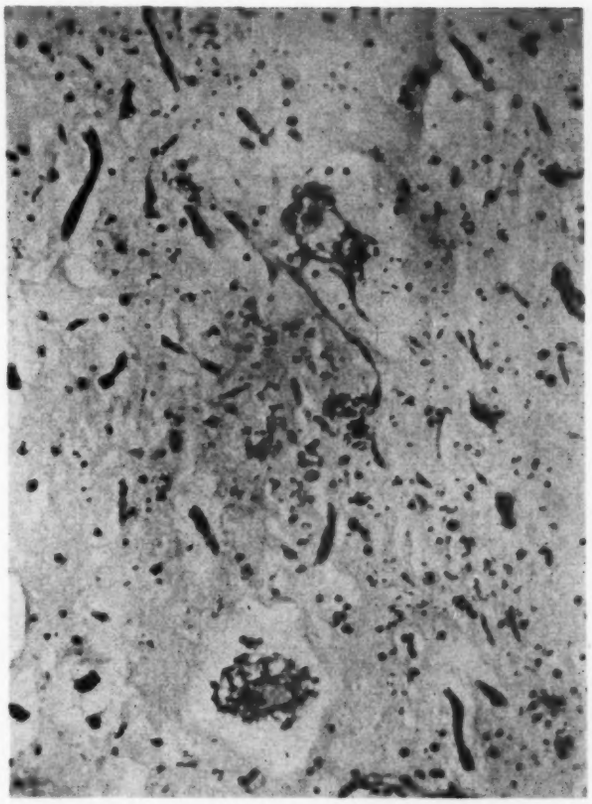

FIG. 12. Case v. Low power photomicrograph showing many capillaries lying in a homogeneous matrix simulating an organising thrombus. Two of the capillaries are surrounded by thin cuffs of loose "myxomatous tissue." (Hematoxylin and eosin.)

tapes were passed about the superior and inferior venae cavae, and around the aorta distal to the subclavian artery. An arterial transfusion was started in the left internal mammary artery. The tapes were tightened about the superior and inferior venae cavae, and a few seconds later about the descending aorta, and the arterial transfusion of oxygenated blood was begun. The incision in the left auricle was enlarged to about 2 inches and opened with the retraction sutures. The tumour was freed from its insertion in the atrial septum with the finger and sponge forceps, and removed in two pieces. No residual tumour could be seen or palpated. The atrium was filled with normal saline to prevent air embolism and the angulated clamp was reapplied to the left atrium with the assistance of the retraction sutures. Tapes were released from the venae cavae and the aorta. The circulation had been interrupted for a period of four minutes. The heartbeat, which had been weak but regular, became strong and the blood pressure rose to 140. The incision in the atrium was sutured with interrupted black silk. The chest wound was closed and the sternum sutured, and both sides of the chest were drained.

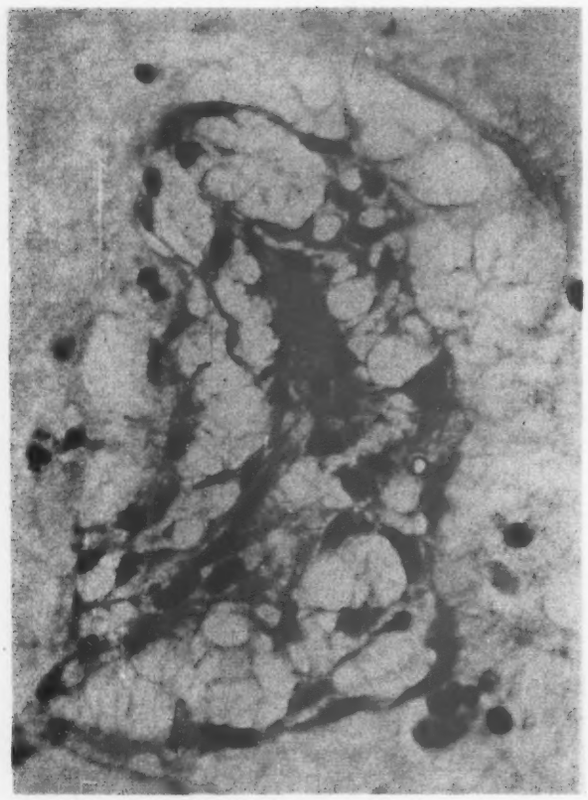

FIG. 13. Case v. High power view of a small focus of myxomatous tissue. (Periodic acid-Schiff stain.)

FIG. 14. Case v. Section stained by the Hale colloidal iron method for acid mucopolysaccharides (connective tissue mucin) which appears gray-black in this photomicrograph. Note the concentration about capillaries corresponding to the cuffs of "myxomatous tissue." This excessive amount of connective tissue mucin is not observed in organizing thrombi.

The patient had a rather stormy convalescence; pneumonia developed in the right lower lobe, which gradually cleared. By September, 1956, she was feeling well, was able to do all her own housework and did not become short of breath on walking several level blocks or up two flights of stairs. However, she continued to have a faint mitral systolic murmur. X-ray films of the chest, showed a decrease in heart size to normal, with almost complete clearing of the lung fields. (Fig. 10.)

The pathologic specimen consisted of two pieces of yellowish, variegated, gelatinous-appearing tissue measuring about 6 cm. in total diameter and with a pedicle about 1 cm. in diameter. (Fig. 11.) The report on the microscopic examination and the conclusion of the pathologist, Dr. H. E. Taylor, follows:

Microscopic examination: multiple sections were stained by hematoxylin and eosin, phosphotungstic acid hematoxylin, periodic acid-Schiff method and Hale's colloidal iron method for acid mucopolysaccharides (connective tissue mucin). In the routine sections much of the tissue architecture was disrupted and obscured by fresh hemorrhage. Many small capillaries occasionally surrounded by thin cuffs of myxomatous tissue were separated by the hemorrhage. Elsewhere islands of homogeneous, mucoid matrix were identified in which lay similar capillaries and scattered plump fibroblasts. (Fig. 12.) Here again, cuffs of myxomatous tissue surrounded occasional capillaries. (Fig. 13.) The homogeneous mucoid matrix and the pericapillary cuffs of tissue were both strongly positive for acid mucopolysaccharides in the slides stained with colloidal iron. (Fig. 14.) The same material did not react with the P.A.S. method.

Conclusion: the specimen was a primary myxoma of the heart with a high connective tissue mucin content (so-called mucinous degeneration of a myxoma). Because the hemorrhagic areas simulated to some extent the early organization of a thrombus, a number of thrombi in various stages of organization from both human and experimental animal material were examined by the same methods noted previously. In no instance was there any accumulation of acid mucopolysaccharides other than the usual small amounts associated with immature fibroblasts. These findings in no way compared to the excessive amounts noted in the heart tumour.

SUMMARY AND CONCLUSION

1. Five primary tumours of the heart which were treated surgically are reported. Two patients are apparently cured, one was improved for several years and two died.

2. Cardiac tumours may be diagnosed preoperatively when they are suspected.

3. When removing tumours from the surface of the heart, great care must be exercised to prevent injury to the coronary artery and the aorta to which they may be firmly attached.

4. Endocardial tumours should be removed under vision with the aid of hypothermia or a heart-lung apparatus.

5. The differentiation between an organizing atrial thrombus and myxoma has proved difficult in some instances, so that many myxomas may have been missed in the past. A method is described in which the demonstration of ground substance in the lesion has proved to be valuable in making such a differentiation.

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DISCUSSION

Lyman A. Brewer, III (Los Angeles, Calif.): Dr. Robertson has presented an exceedingly instructive and well treated series of cardiac tumors. Primary benign neoplasms of the pericardium in my experience have been mainly pericardial cysts. Of a series of 124 operated cases of mediastinal tumors, eleven proved to have the diagnosis of cysts of the pericardium. Although these cysts may be asymptomatic or produce only mild symptoms, their removal is strongly indicated since they are indistinguishable from carcinoma of the lung or potentially malignant tumors.

Malignant neoplasms of the pericardium are, for the most part, metastatic lesions. They are not rare; in fact 12 per cent of advanced cases of bronchogenic carcinoma invade the pericardium. A severe and chronic type of cardiac tamponade is produced which is very disabling. We have been pleased with the "pericardial-pleural window" operation that this discusser presented before the Association three years ago. This procedure relieves the tamponade and offers effective palliation. It is gratifying that Dr. Robertson has pointed out benefits of this operative technic, for one finds little reference to it in the medical literature.

Primary tumors of the myocardium and endocardium are much more common than metastatic neoplasms of these sites (20:1). I have recently reviewed the cases of primary myocardial tumors at the Los Angeles County General Hospital and the Tumor Registry of the Cancer Commission,

California Medical Association, at this same institution, and the White Memorial Hospital. Of eighteen primary neoplasms of the heart, approximately one-half were sarcomas while the remainder were benign. Benign tumors included five myxomas, one fibroma and one lipoma. In retrospect, two or more of these cases were probably operable. The myxomas, as reported by the author, arose in the left auricle and produced obstruction to the mitral valve in all but one instance. This patient, a fifty year old gardener, gave a two-year history of substernal pain and syncope when he bent forward to pick up an object from the ground. A presystolic and systolic murmur were inconstantly heard near the apex. These findings suggested rheumatic heart disease with possible mitral and aortic involvement although the cardiac silhouette was not appreciably enlarged on the roentgenogram. The patient died suddenly while bending over at work. Autopsy revealed a myxoma of the left atrium extending by a pedicle into the left ventricle. This tumor, which was 4 by 6 cm., dangled free in the ventricle when the patient was erect but fell anteriorly to obstruct the aortic valve when the patient bent forward. This case was possibly operable using a technic similar to that reported by Dr. Robertson, to be drawn upward through the mitral valve.

Dr. Robertson is quite correct in recommending hypothermia or heart-lung apparatus when attempting to remove a myxoma of the atrium. There were eight unsuccessful attempts to remove such a tumor, as compared to four successful attempts (Crafoord, Bigelow, Nichols and Scannell) besides the author's. To be sure, Nichols has devised an ingenious snare and "landing net" to remove these growths by closed technic, yet the open method is more thorough and appears to offer less chance of embolism.

Encouraged by these successful excisions of the intracardiac myxomas, more serious attempts should be made for clinical diagnosis. All cases that clinically appear to have mitral or tricuspid stenosis must be considered as possible atrial tumor suspects, particularly in the absence of rheumatic fever or when the tricuspid is involved singly. Inconstant and positional murmurs pointing to a change in mechanical valve obstruction, such as that from a moveable tumor, are important. Blacking out and failure to tolerate certain positions is a most important sign. Finally, direct evidence of a space-filling defect of the cardiac chamber, as seen with the contrast angiocardiogram, points to the diagnosis of an intracardiac tumor just as surely as does barium in the gastrointestinal tract. Of course the shadows produced by irregular bulges of the cardiac silhouette along the mediastinum and evidences of pericardial fluid and cardiac tamponade are further signs of neoplasia of the pericardium or epicardium.

Great care must be exercised when operating upon an intracardiac tumor to take into consideration positional blackouts caused by the tumor, for to operate upon the patient in an unfavorable position has been reported in the literature as resulting in disaster. It seems reasonable to us that the operation may have to be carried out from some most unorthodox approach, such as the sitting-up position, etc. At the present time surgical efforts to excise cardiac tumors have been curative only in benign lesions. In at least one of the cases of sarcoma of the myocardium which have been reviewed, surgical excision might have been performed. With the rapidly advancing progress of cardiac surgery, one cannot exclude the further extension of the current surgical procedures to include excision of malignant lesions of the heart.

Dr. Robertson is to be congratulated for bringing this subject so forcefully to our attention with his fine series of cases, and particularly for the brilliant results in the case of the intracardiac myxoma.

David J. Dugan (Oakland, Calif.): I am very grateful for this opportunity of being able to get a curbstone consultation from Dr. Robertson.

At the present time we have a case which I think might be of interest, and I am interested in Dr. Robertson's report of a case of myxoma that was satisfactorily removed.

Recently I had occasion to see a patient who had the typical signs of mitral stenosis described so well by Dr. Robertson in his case. This woman was fifty-four years of age and lacked the one additional symptom of intermittent fainting. We did not obtain that history before or after surgery.

The woman was operated upon and the mitral area was explored with the preoperative diagnosis of mitral stenosis. Much to my amazement, upon opening the pericardium the auricle was not enlarged; rather it was contracted. There were about 75 cc. of clear fluid within the pericardial sac.

On exploring the left auricle, to my surprise the mitral valve was very widely opened, but just superiorly located and medially there was a mushroom type of tumor mass which I first interpreted as a clot. However, the mass had a very wide pedicle, I think probably 2 inches in diameter, and it floated around in the auricle rather fascinatingly.

The feeling at that time was that the mass was probably a tumor, and the quickest thing we could think to do was to get out of the area.

The patient ran a rather stormy postoperative course, and it is now about two weeks after surgery. I failed to mention this, but she had had a history of emboli prior to this episode, and that was one of the main reasons for the intended commissurotomy.

In reviewing one of the embolic lesions in the left femoral, which was removed by embolectomy, the slides showed definite tumor cells and the pathologist was convinced that it was a myxoma.

We plan to re-explore this patient in the near future under hypothermia, and I will be interested to hear the exact technic used by Dr. Robertson.

Inasmuch as I feel this is not a very short or narrow pedicle, I think that because of the size of the auricle and the size of the pedicle, the flatness of it, we will probably have to remove part of the auricular wall in removing the tumor.

Ross Robertson (closing): I would first like to thank Dr. Brewer for his kind remarks and for filling in some of the gaps in my paper.

Dr. Dugan's case is certainly very similar to ours. Replying to his question regarding the operative technic, I did not get time to say that we used hypothermia. Tapes were placed around the superior and inferior venae cavae, and also around the aorta distal to the subclavian axillary. Since the auricular appendage had been amputated at a previous operation, we had the problem of how to get into this auricle. We used a method that I have not seen described before, which worked very well.

Two rows of transfixion sutures were inserted into the auricle, about four or five on either side. With traction on the sutures a large clamp was readily applied to the auricle outside the rows of sutures. An incision about ¾ inch long was then made between the two rows, and the right index finger inserted as the clamp was released. The tumour was located as at the previous operation and found to have a pedicle about ¾ inch wide on the auricular septum. The finger was withdrawn as the clamp was reapplied.

We then tightened the tapes on the superior and inferior venae cavae and the aorta, and started our arterial transfusion of oxygenated blood. The incision was then extended about 2 inches, blood was sucked out of the auricle and the tumour was removed with the finger and sponge forceps. It came out in two pieces, stripping off the auricular septum cleanly and easily.

The clamp was reapplied, the circulation having been shut off for a period of four minutes. The heart resumed beating strongly. The incision was closed in the usual way without difficulty and the patient made a good recovery.

The Surgical Treatment of Esophageal Hiatus Hernia

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THE recognition of esophageal hiatus hernia as a cause of upper abdominal distress has evolved mainly during the past two decades. The symptoms are many and confusing, and at times present distinct problems in management. We have reviewed 130 cases of esophageal hiatus hernia treated at the University of California Hospital during the twenty-year period from 1936 to 1956 in an attempt to clarify some of the varied problems caused by this disorder.

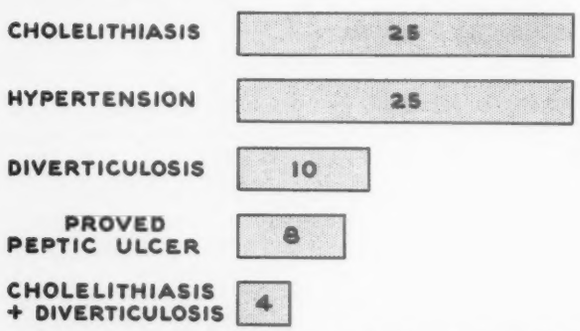

FIG. 1. Concomitant diagnosis. Coexisting disease states in 113 cases of esophageal hiatus hernia.

An acquired short esophagus was present in eleven of the 130 patients studied, and six patients in the series had a congenital short esophagus with thoracic stomach. Since these disorders represented problems which were quite distinct from those of the usual esophageal hiatus hernia, attention was directed toward the remaining 113 patients who were treated surgically for otherwise uncomplicated hiatus hernias.

Women outnumbered men in this series by a ratio of approximately 2:1. The youngest patient was twenty-eight years of age and the oldest was seventy-seven. In the women, the highest incidence of hernia was between the ages of fifty and seventy. In the men a wider spread of incidence over several decades of life was noted, although a significant percentage of hernias occurred in men in the fifth decade of life.

It is interesting to note that in the earlier part of the twenty-year period under study, patients were often treated for such disorders as "ulcer, pylorospasm or gallbladder disease" for several months or years before the correct diagnosis was finally reached. In recent years a prompt, definitive diagnosis and proper treatment are the rule rather than the exception. In many patients an additional major disease was actually present. (Fig. 1.) Cholelithiasis and hypertension were the disorders that most commonly occurred in conjunction with esophageal hiatus hernias. Cholelithiasis was present in twenty-five patients, and hypertension in an additional twenty-five. Diverticulosis was noted in ten patients (8.8 per cent) and peptic ulcer (confirmed at operation or by roentgenologic studies) was present in eight patients (7.1 per cent). Many patients, however, were justifiably treated for an ulcer before its presence was actually confirmed, since their symptoms definitely suggested this diagnosis. The coexistence of cholelithiasis and diverticulosis with esophageal hiatus hernias, sometimes spoken of in terms of a frequently occurring triad, was seen in only four patients in this series (3.5 per cent).

The possibility that coexisting diseases may be the major cause of symptoms rather than the definitely demonstrated hiatus hernia is of paramount importance in the assessment of these problems. It is imperative that these associated diseases be carefully evaluated and, if reasonable evidence exists suggesting that they are in some measure responsible for the

patient's symptoms, treatment of these disorders should be undertaken prior to surgical repair of the hernia. In some instances it is necessary to undertake surgical repair of the hernia and an associated disorder at the same time. This is especially true when cholelithiasis is present in addition to a hernia or when there is a possibility of peptic ulceration.

TABLE 1
TYPE OF ESOPHAGEAL HIATUS HERNIA PRESENT IN 130 CASES

Esophageal hernia	113
Paraesophageal hernia	16
Sliding	95
Combined	2
Acquired short	11
Congenital short	6

Hernias through the esophageal hiatus of the diaphragm are of two main types, paraesophageal and sliding. In the series of 113 cases studied in detail the paraesophageal type was present in sixteen and the sliding type of hernia in ninety-five. A combination of the two types occurred only twice. (Table 1.) The proportion of sliding to paraesophageal hernias in this series was in the ratio of approximately 6:1.

The symptoms that may be produced by an esophageal hiatus hernia vary considerably in type and severity. The majority of the symptoms are the result of incompetency of the sphincteric mechanisms, which allows reflux of gastric contents into the lower esophagus, producing irritation or inflammation of varying intensity. The degree of inflammatory response depends on a variety of factors such as the frequency of regurgitation, the degree of acid and peptic activity of the fluid retrogradely bathing the lower esophagus, and the duration of contact of gastric contents with the esophageal mucosa.

Such distressing symptoms as recurrent nausea and vomiting after eating and epigastric or substernal distress of a burning nature are commonly described, especially by patients in whom the sliding type of esophageal hiatus hernia is present. In severe instances, spontaneous or forced vomiting may be the only action that regularly affords relief. Almost all of the patients in the series complained of nausea, but spontaneous or forced vomiting was mentioned by only 23 per cent.

If much of the stomach lies within the thoracic cavity (as in the paraesophageal hernia), distention of the gastric pouch after eating and upward displacement of the gastric air bubble may cause symptoms of pressure as well as a feeling of heart consciousness. The latter may vary from recognizably irregular heartbeats to rapid and sustained tachycardia, continuing until gastric distention is reduced, either by eructation of gas, vomiting of ingested food or the normal emptying of the stomach.

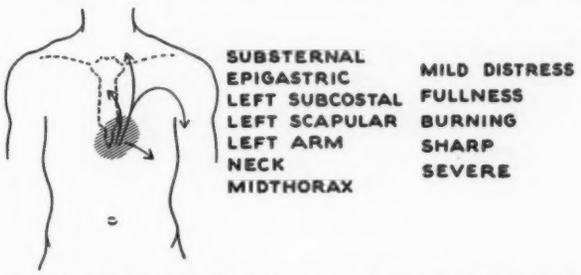

FIG. 2. Analysis of pain (non-positional in thirty-eight cases) in esophageal hiatus hernia.

Pain of varying degrees of severity was the most common complaint of the patients in this series. (Fig. 2.) Substernal and epigastric distress frequently extended to the left costal margin or into the region of the tip of the left scapula, much in the same manner that referred pain in gallbladder disease extends to a similar area on the right side. Occasionally extension of pain down the left arm was noted.

Symptoms of esophagitis occur infrequently in patients with paraesophageal hernias but are common in patients with sliding hernias. Often, rather severe substernal or epigastric burning pain may exist intermittently for years without a demonstrable inflammatory reaction in the lower esophagus. In such instances some degree of chemical irritation exists which momentarily produces symptoms but is not sufficiently prolonged to produce organic changes. Such symptomatic episodes are comparable to the transient "heartburn" experienced by normal persons who have competent cardioesophageal sphincter mechanisms.

The patients in whom esophagitis develops are those in whom the lower esophageal segments are bathed frequently, or perhaps almost constantly, by the acid-peptic contents of the stomach. In such circumstances a recognizable inflammatory process is produced in the lower esophagus. This inflammation is at first limited to the mucosa, which appears as a swollen and redundant membrane containing scattered

white patches of fibrinous material resembling leukoplakia. The mucous membrane between the fibrinous areas is pinker than normal and bleeds easily when touched by instruments. In view of the long history of regurgitation often obtained from patients with hiatus hernias, it

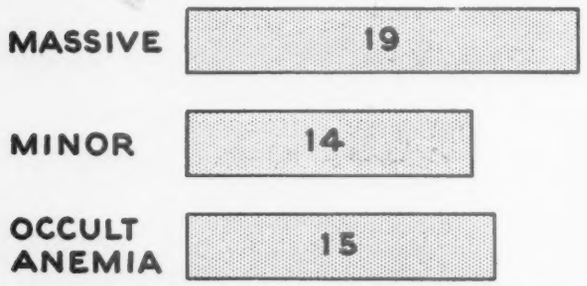

FIG. 3. Forty-eight of the 113 patients exhibited some degree of hemorrhage. The incidence of massive hemorrhage was considerable in this series.

is surprising that the sensitive esophageal mucosa often can resist acid-peptic digestion for long periods of time before penetration into the deeper layers occurs.

In more severe esophagitis the mucosa is redder and contains ulcerations varying in size from 1 to 3 mm. which bleed with the slightest trauma. The lumen may be narrowed as a result of the edematous mucosa and submucosa. Scarring occurs in the late stages of the inflammatory process, causing contraction and narrowing of the lower esophagus. Some degree of fixation of the area is also likely to occur as a result of the accompanying periesophagitis. It is in a circumstance such as this that dysphagia becomes a predominant symptom.

The pain associated with esophageal hiatus hernia is largely due to severe persistent or recurrent esophagitis or to the less common episodes of incarceration. Occasionally the symptoms are described as distressing or bothersome rather than painful. In our series, however, some degree of pain was mentioned by ninety-eight patients, or 87 per cent. In six patients lower thoracic and upper lumbar aching pain was described as the only symptom associated with a demonstrable esophageal hiatus hernia.

Dysphagia was a relatively infrequent symptom in patients in this series, although twenty-eight patients (25 per cent) complained of an occasional sensation of food lodging momentarily in the substernal area. Dysphagia in the absence of organic fibrous stenosis is the result of the piling up of the mucous membrane in an accordion-like fashion because of the physiologically shortened lower esophagus. For this reason dysphagia, if present, occurs in patients with sliding hernias but is rare in patients with the paraesophageal type. The presence of inflammatory edema also helps to produce this type of dysphagia.

Narrowing of the lower esophagus, demonstrated either by roentgenologic studies or by esophagoscopy, was present in only nine patients, or 8 per cent of those patients undergoing definitive surgical treatment for esophageal hiatus hernias of the paraesophageal and sliding types. The presence of any significant degree of organic narrowing was uncommon in long-standing cases of hiatus hernia, even in those which were completely neglected.

Hemorrhage, ranging in severity from occult anemia to episodes of massive bleeding, was noted in forty-eight patients or 42.4 per cent. In seven of these patients esophagoscopy produced no evidence of esophagitis, which lends some support to the thesis (presented in detail in a later section of this article) that the inferior esophageal constrictor may play a part in the prevention of esophagitis. That massive hemorrhage can occur in patients with hiatus hernias cannot be denied. Evidence of hematemesis or melena of considerable severity was found in nineteen patients (16.8 per cent). Hemoglobin values as low as 6 to 8 gm. were recorded in many of these patients on hospital entry. The incidence of gross hemorrhage in our series was greater than that in most of the cases reported in the literature. Minor degrees of hemorrhage, however, were noted more often in the present series than were major episodes of bleeding. (Fig. 3.)

The formation of paraesophageal and sliding hernias (and the symptoms they produce) depends on the disturbances in the normal anatomy and physiology in the area of the esophageal hiatus of the diaphragm. Of importance in the production of hiatus hernias are the fibroareolar attachments at the esophageal hiatus and the complex sphincteric mechanisms which ordinarily prevent regurgitation.

In the normal state the abdominal portion of the esophagus is maintained in its regular position in part by the peritoneum, which invests the stomach and abdominal esophagus and reflects itself onto the undersurface of the diaphragm. That portion between the esophagu

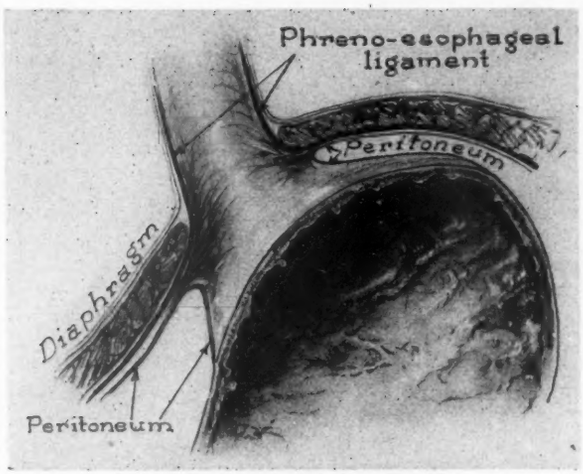

FIG. 4. The normal anatomy in the region of the esophageal hiatus. The phrenoesophageal ligament is a strong band of fibrous tissue which plays a major role in maintaining the abdominal esophagus in place.

and the diaphragm forms somewhat of a bridging ligament and helps to hold the abdominal esophagus in its normal position.

The diaphragmatic (phreno)esophageal ligament is also an important structure in the maintenance of the cardioesophageal junction in its normal position. This ligament is formed when the muscular fascia of the undersurface of the diaphragm is reflected upward along the esophagus and attaches itself to the circular muscular fibers of the esophagus at the site of the inferior esophageal constrictor. As such, this ligament acts as the antagonist of the longitudinal muscle of the esophagus and prevents the elastic esophagus from pulling the stomach into the chest during swallowing. When the esophageal hiatus is of normal caliber, these fibroareolar attachments are closely applied to the lower esophagus and diaphragm as tubular sheets of strong fascial tissue, surrounding the abdominal esophagus and holding this area firmly in place. (Fig. 4.)

The physiology of the sphincteric mechanisms is also of extreme interest. The question of the role of the diaphragm in preventing regurgitation is a controversial one. That it does play some part is agreed with by most observers, certainly by those who have had the opportunity of inserting an examining finger upward into the esophagus through a gastrostomy opening. Under these circumstances no one can deny the presence of muscular contraction as exerted upon the examining finger. In the normal person, and perhaps in the patient with a hiatus hernia, some diaphragmatic pinchcock action exists. Below this diaphragmatic muscle-action area the cardioesophageal junction itself is an important factor in preventing regurgitation.

If the cardioesophageal junction remains below the diaphragm, the normal angle at

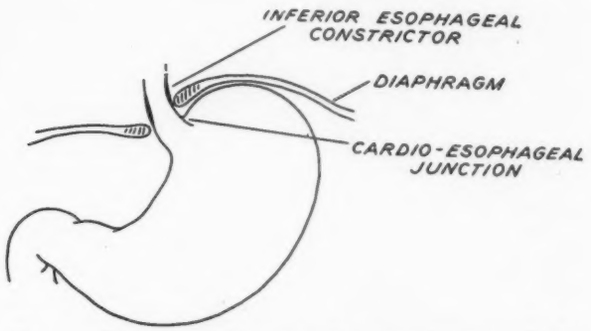

FIG. 5. The normal anatomic arrangement of the sphincteric mechanisms. The inferior esophageal constrictor, the diaphragmatic pinchcock and the cardioesophageal junction constitute a three-in-line sphincter group that prevents regurgitation.

which the esophagus enters the stomach is maintained. The preservation of this anatomic relationship is of paramount importance; it is believed that at this oblique line of entry the interplay of the musculature of the lower esophgus and the circular and oblique fibers of the stomach accounts for the competency of the cardioesophageal junction. A true sphincter muscle has never been shown to exist, although a physiologic sphincteric mechanism is present in the normal person. Above the level of the esophageal hiatus of the diaphragm, the inferior esophageal constrictor may also play a part in preventing regurgitation. In the normal person, therefore, the sphincteric mechanisms form a sort of three-in-line defense against gastric reflux. (Fig. 5.)

The aberrations which occur in these normal anatomic and physiologic relationships provide the basis for the formation and symptomatology of the two types of esophageal hernias. In the *paraesophageal hernia* the esophagus is of normal length. Its abdominal portion is held firmly in place by its fibroareolar attachments, in spite of the enlargement and weakening of the esophageal hiatus, and the cardioesophageal sphincter mechanism remains undisturbed. Since it is in its normal position, the esophagus remains competent for the most part and allows food to pass from the esophagus into the stom-

FIG. 6. A pad of fat developing in the bare area posteriorly may serve to weaken and dilate the diaphragmatic crura.

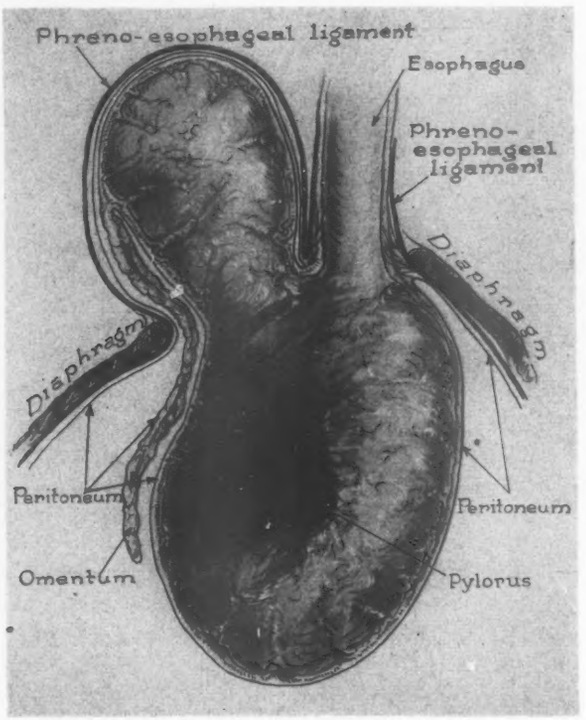

FIG. 7. Cross sectional drawing of the paraesophageal hiatus hernia. The esophagus is of normal length and the cardioesophageal angle is undisturbed. The sac may be preformed or can result from weakening of a segment of the phrenoesophageal ligament.

ach in an orderly manner, thus preventing regurgitation.

The peritoneal investment provides the abdominal esophagus and stomach with a complete serous coat, except at the reflection of the omenta at the curvatures and in a small area at the left of the cardia that is in direct contact with the diaphragmatic musculature of the crural canal. This bare area in contact with retroperitoneal tissue acts as a two-edged sword in the development of either type of hiatus hernia. On the one hand, it serves as a relatively fixed point of the cardia of the stomach, since the reflection must be continuous with the relatively immobile posterior peritoneum. This prevents upward herniation of the posterior wall of the stomach in spite of an enlarged esophageal hiatus. On the other hand, an accumulation of fat in this bare area may serve to weaken the crura and allow them to lengthen and enlarge, thus setting the stage for herniation. (Fig. 6.) If the posterior bare area is relatively immobile, the paraesophageal hernia can form only when a segment of the gastric fundus rolls up anteriorly or laterally and the posterior aspect of the stomach remains in a normal position.

In the paraesophageal hernia the stomach can be herniated into a preformed sac of peritoneum remaining in the thorax after embryonal descent of the stomach, or by the lengthening and weakening of a segment of the circumferential fibroareolar attachments of the lower esophagus. In the latter case the elongated peritoneal reflection between the esophagus and diaphragm is carried upward. In both instances a peritoneal hernial sac will exist in the paraesophageal type. (Fig. 7.)

Regurgitation usually does not occur with any greater frequency among patients with paraesophageal hernias than it does in normal persons, since the location and integrity of the cardioesophageal junction remain largely undisturbed. Absence of regurgitation in this type of hernia is, of course, not absolute. Occasionally the bulk of the herniated stomach displaces or causes upward traction of the cardioesophageal junction, disturbing its function and allowing regurgitation to occur.

In our group of eighteen cases of paraeso-

phageal hiatus hernias (which includes two cases of paraesophageal hernias combined with the sliding type) eleven patients had a history of pyrosis, which suggested regurgitation. In none of these, however, was esophagitis present, so that the frequency of regurgitation must have been minimal. Esophagitis is not a bothersome problem in this type of hernia. On the contrary, the symptoms are largely those produced by distention of the herniated viscus and pressure upon adjacent organs. As a result, patients with paraesophageal hernias may complain of a feeling of fullness after meals, anterior pressure within the left thorax, palpitation, shortness of breath, nausea and vomiting, and pain of varying degrees of severity. The pressure upon adjacent viscera frequently produces dyspnea and angina-like pain. This was true in five of our patients. The latter complaint often presents a considerable problem in diagnosis, since this particular type of pain may be confused with that produced by disease of the coronary arteries.

The *sliding hernia*, with respect to the symptoms produced and the possible etiologic mechanisms involved, presents quite a different problem. Here, as in the paraesophageal hernia, the crural canal is enlarged and its fibers weakened, thinned out and elongated. However, in the sliding hernia the attachments that normally tether the abdominal esophagus to its normal position are compromised and allow the cardioesophageal junction to be displaced upward into the posterior mediastinum. (Fig. 8.)

The weakening of the normal attachments is only part of the etiologic background. The enlarged hiatus may be either congenital in origin or related to the enlargement incident to advancing age. The possibility also exists that the hiatus may weaken and enlarge as a result of accumulated fat in this area. Pressure immediately below the esophageal hiatus is increased by such actions as bending forward, acute flexing of the knees and coughing. If fat accumulates in the area of the crural canal, it is not difficult to visualize how the increased abdominal pressure may force the leading edge of the fat upward, causing enlargement of the hiatus. The increased abdominal pressure that results from obesity plays a part in forming the hiatus hernia, or at least helps to perpetuate symptoms once the hernia is formed. In our series 64 per cent of the patients who were operated upon were moderately to markedly obese and 36 per cent were of relatively normal weight.

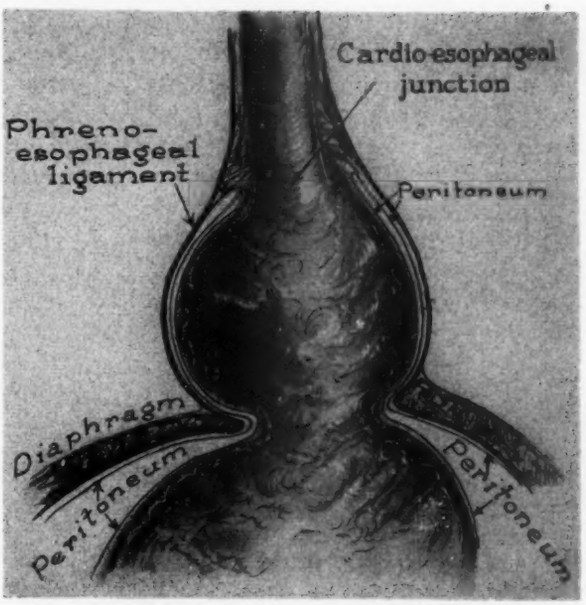

FIG. 8. The sliding type of esophageal hiatus hernia. Note the attenuated phrenoesophageal ligament and the peritoneum folded upon itself as it is carried upward. The cardioesophageal angle is disturbed. The esophagus enters the herniated pouch without angulation at the apex.

Weakening and elongation of the ligamentous structures and enlargement of the hiatus combine with two other factors to produce the sliding hernia: positive pressure in the abdomen and negative pressure in the thorax tend to cause a one-way transfer of the lower esophagus and stomach upward into the posterior mediastinum and, in addition, the normal esophagus has considerable elasticity and tends to pull the gastroesophageal area upward.

As a result of these several factors, which may operate independently or in combination, the entire unit of the lower esophagus and upper stomach comes to lie in the posterior mediastinum. This position usually is not permanent. The structures are not fixed except in long-standing and neglected sliding hernias in which submucous and periesophageal scarring and fibrosis cause adherence to the surrounding area and fix the gastroesophageal region rather rigidly in the herniated position. The unit usually can be reduced spontaneously, however, when the patient assumes an erect position, by the weight of food in the stomach or by surgical means. The esophagus is only physiologically shortened under these conditions, and through

traction (by means of the erect position or surgical correction) it can again assume its normal length and allow the cardioesophageal junction to be placed below the diaphragm. As long as the cardioesophageal junction is displaced, it remains compromised and allows regurgitation

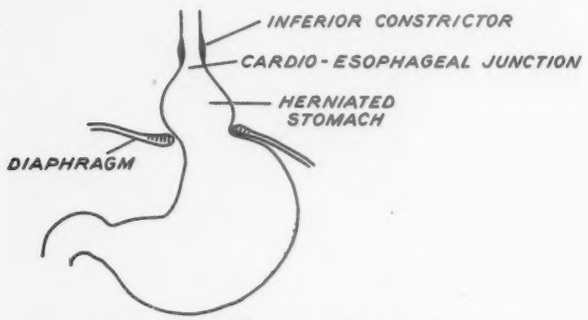

FIG. 9. The interaction of the three-in-line sphincter mechanisms, some acting normally and others abnormally, may account for some of the signs and symptoms of esophageal hiatus hernias.

to occur, thus producing the main symptom of such hernias.

When the hiatus becomes enlarged (mainly in the anteroposterior diameter), the phrenoesophageal ligament must of necessity weaken and elongate as the hiatus enlarges. Thus, instead of remaining as a short band of strong tissue between the esophagus and crural margins, the ligament becomes a broad weakened band of tissue that can no longer hold the cardioesophageal mechanism in place. As a result the tethering ropes are loosened, the antagonistic effects of positive abdominal pressure and negative intrathoracic pressure are no longer combatted effectively, and the entire unit of the lower esophagus and stomach slides up into the chest.

The peritoneal reflection from the stomach onto the diaphragm also shares in this migration, so that a pseudosac of peritoneum also will be part of the unit. It is not a true sac; it does not end blindly but terminates at its uppermost point (the transposed gastroesophageal junction) by folding upon itself. In this fashion it appears as a double-lined space surrounding the herniated stomach and lower esophagus.

It seems reasonable to assume that the initial enlargement of the hiatus is in the anteroposterior plane, since the crural fibers are suspended in this direction. Any weakening of the ligaments, therefore, would cause lengthening of the individual muscle strands which in turn would produce enlargement, mainly in the anteroposterior diameter. Similarly, the weakening of the phrenoesophageal ligament must occur in the same plane. When this takes place, the stage is set for the positive intra-abdominal pressure to stretch the ligament further, slowly but relentlessly, so that the entire circumference of the ligament is eventually comprised, allowing an unhindered upward protrusion of the gastroesophageal segment.

Although symptoms produced by pressure may be present in patients with sliding hiatus hernias (especially if the herniated portion of the stomach is large), more often the symptoms are caused by regurgitation of gastric contents into the lower esophagus. In the ninety-five cases of sliding hiatus hernias in our series, evidence of episodes of regurgitation (indicated by a history of heartburn which usually became worse upon lying down) was present in almost every case.

The symptoms and signs of sliding hiatus hernias are often confusing, and as such are subject to a wide variety of interpretations. Many patients complain of regurgitation, pyrosis and substernal distress, suggesting the presence of esophagitis, although little or no inflammation is actually present. In our series no evidence of esophagitis was present in eight of the fifty-four patients who underwent preoperative diagnostic esophagoscopy, and in an additional thirteen patients only a minimal inflammatory reaction was noted.

The concept of normal or abnormal action of the three-in-line valve mechanisms in the region of the diaphragmatic hiatus offers an explanation of some of the confusing signs and symptoms seen in the patient with a sliding esophageal hiatus hernia. (Fig. 9.) In the patient who has evidence of regurgitation without esophagitis it can be postulated that the inferior esophageal constrictor remains strongly competent and prevents reflux in spite of the enlarged esophageal hiatus and the obviously disturbed cardioesophageal junction. Reflux esophagitis is undoubtedly present to some degree in patients who have a weak inferior constrictor. Moreover, if a relatively strong diaphragmatic pinchcock mechanism still exists, contraction upon the herniated stomach might well force its contents upward into the esophagus in spite of the strength of the inferior constrictor.

The presence of a strong inferior constrictor and a still functioning diaphragmatic pinchcock might also explain another confusing sign seen in sliding esophageal hernias, namely, gross or microscopic evidence of hemorrhage without evidence of esophagitis. Hemorrhage is a common finding in esophagitis and, although often minimal, occasionally may be alarming. In the absence of esophagitis, hemorrhage is usually attributed to congestion in the temporarily incarcerated stomach and the bleeding is presumed to be due to poor venous drainage.

A functioning inferior constrictor above the herniated pouch may prevent esophagitis in patients with the sliding type of hernia. The constrictor and the diaphragmatic pinchcock may form the two ends of a functioning unit of muscle, enclosing the herniated portion of the stomach between them. This would allow gastric contents to act continuously upon mucosa already partly damaged by venous congestion. The focal peptic gastritis may produce considerable hemorrhage entirely from the stomach without evidence of esophagitis.

The concept of normal or abnormal action of the several valve mechanisms also serves to explain how symptoms can be produced regardless of the size of the hernia. Even a small hernia may produce severe symptoms of esophagitis if regurgitation occurs as the result of the incompetence of the valve mechanisms. We believe that the size of the hernia is not an important factor in making the decision to operate, in spite of the fact that 100 of the 113 hernias in this series were moderate to large in size and the remaining thirteen were small. The symptoms of an esophageal hiatus hernia are due to regurgitation, which is not dependent on the size of the hernia but on a derangement in the various sphincter mechanisms.

Such a concept can also explain the relative lack of symptoms in patients with very large hernias. The completely asymptomatic hiatus hernia can also be explained on this basis. If the diaphragmatic pinchcock is patulous and without contractile strength and if the inferior constrictor is strong, neither esophagitis nor retention of gastric secretions occurs. In such instances a portion of the stomach is merely displaced but otherwise functions normally. (Fig. 10.)

The technic of surgical repair of sliding hiatus hernias is also related to the concept of valvular mechanisms. In the surgical treatment, two of the three-in-line sphincter mechanisms are successfully restored by the replacement of the cardioesophageal junction below the diaphragm and partial closure of the esophageal hiatus. Thus the normal angle of entry of the esophagus into the stomach is re-established and maintained.

FIG. 10. This patient, under treatment for a tuberculoma of the right upper lobe, had a large, completely asymptomatic esophageal hiatus hernia. Note the outline of the herniated stomach.

If the crura are lightly approximated posteriorly,* where the greatest attenuation has occurred, the diaphragmatic pinchcock mechanism is restored and the sphincteric action helps to prevent regurgitation. Anteriorly, the decussation of the crura remains strong and provides a well rounded sling for the esophagus, even in the most severe cases of hernia. For this reason the crura should be approximated only in the posterior portion of the crural canal. Moreover, since laterally placed sutures would interfere with the contraction of crural fibers suspended in the anterioposterior direction from the lumbar vertebrae, there should be no lateral or medial suturing along the extent of either crus.

Replacement of the cardioesophageal junction below the diaphragm and its fixation there (by tethering the attenuated phrenoesophageal

* One of us (H. B. S.) occasionally approximates the crura anteriorly as well as posteriorly.

ligament circumferentially around the esophageal orifice) serves to restore the normal anatomy completely. The surgically shortened phrenoesophageal ligament may again work in unison with the inferior constrictor and provide a further barrier to reflux esophagitis.

TABLE II
OPERATIVE PROCEDURES

Transthoracic Approach (84)	
Reduction only	36
Diaphragm opened, reduction and suture of ligament to diaphragm	40
Hiatus advanced	7
Gastroesophageal resection	1
Abdominal Approach (29)	
Suspected biliary or gastric disease	8
Cholecystectomy	5
Gastric resection	1
Vagotomy	1
Pyloroplasty	1
	16

RESULTS OF SURGICAL REPAIR

The abdominal approach was used in twenty-nine of the surgically treated cases, and the transthoracic approach in the remaining eighty-four. The abdominal approach was the technic of choice in sixteen cases because of suspected or previously demonstrated gallbladder disease or gastric disorder. In half of these sixteen patients no biliary or gastric disease was found; in the remaining eight, resectional surgery was performed in addition to repair of the hernia. (Table II.)

In only one case in which the thoracic approach was used was resection necessary to treat a penetrating esophageal ulcer. The hernia was repaired in the remaining patients by simple reduction and posterior closure of the crura (thirty-six cases), by Allison's technic of reduction and securing of the cardioesophageal junction below the diaphragm by circumferential suturing of the phrenoesophageal ligament to the undersurface of the diaphragm (forty cases), or by advancement of the esophageal hiatus to the dome of the diaphragm (seven cases).

In six patients in whom the transthoracic approach was used the left vagus nerve was believed to be permanently injured during mobilization of the lower esophagus. In none of these patients, however, did any untoward symptoms develop.

In the follow-up period, ranging from three months to sixteen years, the over-all results of surgical repair were good to excellent regardless of the type of surgical procedure or approach used. Good results can usually be expected with either approach or method of repair. The common denominator of all technics is the replacement of the cardioesophageal junction below the diaphragm. When this is accomplished, the sphincter mechanism again becomes competent at this level. The good results reported from repair by advancement of the hiatus to the dome of the diaphragm depend on the reduction of the cardioesophageal junction to its normal site below the diaphragm. If, in addition, the patient possesses a well functioning inferior constrictor, two of the three-in-line mechanisms will be re-established. It should be stressed, however, that if a specific method can restore the normal anatomy, it should be the operation of choice. Allison's technic appears to restore the anatomy more completely and securely than does a simple reduction that ignores the attenuated phrenoesophageal ligament.

TABLE III
RECURRENCES*

No. of Recurrences	Type of Hernia	Repair
Abdominal Route		
4	Sliding	Simple reduction
Thoracic Route		
8	Sliding, 7; paraesophageal, 1	Reduction, 5; suture of ligament, 2; new hiatus, 1

* Only 1 recurrence after original repair took place in the paraesophageal group. The remaining recurrences were in the sliding type.

Hernias recurred in twelve patients of our series after initial repair at the University of California Hospital. (Table III.) Four recurrences took place in patients in whom the abdominal approach was used; all of the hernias were of the sliding type, repaired by simple reduction alone. The thoracic approach resulted in eight recurrences; seven of the hernias were of the sliding and one was of the paraesophageal type. Simple reduction was performed in five,

FIG. 11A. Radiologic studies eight months before entry demonstrated a shadow compatible with a sliding esophageal hiatus hernia, unchanged in contour from that shown in films obtained two years previously.

FIG. 11B. Radiologic studies at the time of hospital admission demonstrated a mass in the herniated portion of the stomach.

Allison's technic in two, and in one the esophageal hiatus was advanced to the dome of the diaphragm.

Four patients in whom the hernia recurred had symptoms of sufficient severity to require a second operation. The secondary repair (accomplished transthoracically in each instance) resulted in successful alleviation of symptoms. The symptoms of the remaining eight patients with recurrent hernias were less severe and control by conservative means was possible. It should be mentioned that only two recurrences were noted when reduction plus circumferential suture of the phrenoesophageal ligament was done, while ten hernias recurred in cases repaired merely by reduction of the hernia followed by closure of the diaphragmatic crura.

In two patients in this series adenocarcinoma developed in the herniated portion of the stomach. Each of the patients had symptoms suggestive of a long-standing esophageal hiatus hernia. Dysphagia occurred late in each instance, signifying the onset of organic obstruction.

One of these patients, a fifty-nine year old man, had a ten-year history of mild epigastric distress with pyrosis. Dysphagia was first noted eight months before entry into the hospital and was accompanied with mild substernal pain. Roentgenologic studies were interpreted to show only an esophageal hiatus hernia of moderate size. (Fig. 11A.) Conservative treatment gave only partial relief of symptoms. An episode of hematemesis occurred two weeks before entry; roentgenologic studies showed a change in the contour of the herniated portion of the stomach. (Fig. 11B.) During esophagoscopy an obstruction was encountered at the cardioesophageal junction. On gross examination it appeared to be a fibrotic stricture. Histologic study of a specimen removed for biopsy, however, showed that the obstruction was due to adenocarcinoma. The presence of malignant tissue in the herniated portion of the stomach was verified at operation.

The second patient, a seventy-six year old man, had a six-year history of epigastric distress after eating, presumably due to regurgitation of gastric contents into the lower esophagus. Hiccoughing after meals developed four months prior to entry into the hospital. Roentgenologic studies showed a hiatus hernia of the sliding type. (Fig. 12A.) Conservative treatment gave no relief, and dysphagia while eating solid foods became evident one month before entry. Esophagoscopy demonstrated a concentrically narrowed area at the cardioesophageal junction, which again was thought to be fibrous in nature. A biopsy was performed which suggested the existence of distorted gastric glands in the

FIG. 12A. Radiologic studies were interpreted as demonstrating a hiatus hernia of the sliding type, with esophagitis and moderate stenosis.

FIG. 12B. Repeat studies at the time of admission to the hospital showed a greater deformity of the herniated stomach and increased obstruction at the cardioesophageal junction.

fibrotic tissue. Roentgenologic studies at the time of admission showed greater deformity of the herniated stomach and more obstruction at the cardioesophageal junction. (Fig. 12B.) A second esophagoscopy and biopsy revealed the presence of gastric adenocarcinoma. During operation, malignant tissue was noted in the herniated portion of the stomach.

It is interesting to speculate on the role that chronic inflammation, resulting from regurgitation of acid contents into the herniated stomach and lower esophagus, played in the ultimate development of malignant disease in these patients. That malignant growth develops elsewhere in the body from a background of chronic inflammation is quite well understood. It is reasonable to assume, therefore, that such long-continued inflammation could play a similar part in the development of gastric cancer in the herniated portion of the stomach.

Four deaths occurred during the immediate postoperative period (3.5 per cent). (Table IV.)

TABLE IV

ANALYSIS OF FATAL CASES

Patient, Sex and Age (yr.)	Reason for Surgery	Approach	Postoperative Death	Cause
L. H., F, 67...	Repeated incarceration	Thoracic	10th day	Septic infarcts of liver, kidneys, spleen
F. J., M, 63...	Pain	Thoracic	2nd day	Diaphragmatic suture line rupture; gastric dilatation, acute
M. Mc., F, 77.	Severe pain, bleeding	Thoracic	12 hr.	?Anesthetic death; ?coronary artery occlusion
C. E., F, 58...	Pain, anemia	Thoracic	5th day	Occlusion of cerebral artery from embolus from femoral vein through foramen ovale

The first of these patients was a sixty-three year old man who died two days after operation from the effects of rupture of the diaphragmatic incision, incarceration and acute dilation of a large portion of the stomach in the left thorax, and attendant cardiopulmonary collapse.

Another patient, a seventy-seven year old woman, died before recovering from anesthesia. This patient had complained of severe pain and recurrent episodes of bleeding, which justified an operation. The third patient, a sixty-seven year old woman, died ten days after transthoracic repair of a sliding hiatus hernia that had been repeatedly incarcerated. An autopsy was performed which revealed multiple septic infarcts whose site of origin could not be determined.

The fourth patient, a fifty-eight year old woman, had had distressing symptoms of epigastric pain and secondary anemia. She died five days postoperatively from the effects of an embolus into the left middle cerebral artery. A left femoral vein thrombosis had occurred; the embolus apparently passed through a patent foramen ovale.

SUMMARY

The surgical treatment of esophageal hiatus hernias is closely related to an understanding of the disturbed anatomy and physiology of the cardioesophageal sphincter mechanism, the diaphragmatic crural opening and the inferior esophageal constrictor.

Emphasis is placed upon the importance of the three-in-line sphincter mechanisms. Their normal function prevents regurgitation and maintains the normal motor function of the lower esophagus. The interplay of these systems, some of which may act normally and others abnormally, may explain many of the heretofore confusing symptoms produced by an esophageal hiatus hernia.

In the absence of disabling disease of the major body systems, operative repair of an esophageal hiatus hernia is justified, for not only are the symptoms produced by either type of hernia distressing, but also the persistent or recurrent inflammation (initially confined to the esophageal mucosa) may eventually lead to submucous fibrosis, ulceration, stricture formation or perhaps even carcinoma. When stricture formation occurs, surgery becomes a necessity and is not only more formidable than the primary repair but also is frequently far less successful.

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DISCUSSION

Carleton Mathewson, Jr. (San Francisco, Calif.): I have read Drs. Grimes and Stephens' paper with a great deal of interest because of my personal interest in this subject and because they have concerned themselves with the analysis of a most perplexing problem that has baffled anatomists, physiologists and surgeons alike since Helvetius first described an anatomic sphincter at the lower end of the esophagus in 1719. Since that time there has been a continued difference of opinion, not only as to whether or not a sphincter exists, but also, if there is such a mechanism, where and how it exerts its effect. The apposed opinions of such careful observers as Lendrum and Lerche, each of whom has dissected more than 100 esophagi, are

typical of the controversy that exists concerning this problem. Lendrum denies that a sphincter exists, while Lerche is convinced of the original description by Helvetius of an anatomic sphincter.

It would seem to me that the controversy as to whether there is or is not an anatomically distinct sphincter at the cardia is less important than the well documented fact that there obviously is a physiologic sphincter mechanism which apparently utilizes certain unspecialized circular muscular segments to cause segmental constrictions near the cardia demonstrable both by esophagoscopic and radiologic observations acceptable to most competent investigators. In addition, it appears that the angle of entrance of the esophagus into the stomach plays a significant role in the prevention of regurgitation. Careful clinical and laboratory studies reported by Marchand, which were substantiated by Cohn and others in our clinic and reported to this Society a year ago, emphasize the great importance of this particular mechanism.

The stopcock effect of the crura of the diaphragm as emphasized by Jackson has been denied by most observers, who believe that the diaphragm plays no coordinate part in the apparent specific functions of the lower esophageal structures.

Drs. Grimes and Stephens have attempted to explain various symptom complexes related to diaphragmatic hernia on the basis of relative competence or incompetence of one or the other of these three mechanisms, assuming that in the normal person all three play an important role in the prevention of regurgitation. I cannot quarrel with the authors because I cannot prove or disprove their contentions. I can, however, substantiate their observations that many very large diaphragmatic hernias, particularly of the paraesophageal type, do not cause symptoms and that frequently the small, unsuspected sliding type hernias produce a variety of untoward symptoms not uncommonly attributed to other diseases.

It also has been our observation that restoration of the cardia to its normal position below the diaphragm after repair of the hernia will restore normal sphincter action. This has not been our experience when other types of repair have been undertaken.

It now is becoming clear that many persons suffering from this condition have been subjected for years to a variety of remedies, such as diets, antispasmodics, antacids and cardiac remedies, without relief. Many of these patients have been cured by the repair of what appeared to have been an insignificant hiatus hernia.

Because of the great difficulties sometimes encountered in the differential diagnosis of this malady and because we have been reluctant to subject a patient to surgical repair of a hiatus hernia unless we are reasonably certain the operation will relieve the symptoms, we have subjected those patients who present a significant diagnostic problem to a short course of pneumoperitoneum.

In a previous study on a group of patients with known symptomatic hiatus hernias we were able to produce prompt and lasting relief of symptoms by a series of injections of air into the abdominal cavity. The same relief was not obtained in a group of patients whose symptoms, although similar, had been proved to be related to other diseases such as angina or chronic cholecystitis.

This method in our hands has proved to be a most useful means of differential diagnosis.

I am sure there is still much to be learned about this most interesting surgical problem and I am grateful to Drs. Grimes and Stephens for this analysis, which brings us closer to a correct solution of the problem.

PAUL C. SAMSON (Oakland, Calif.): I think this is a very excellent paper, and I rise to talk about something that I am sure is mentioned in the paper and has been of considerable interest to me. This is the use of the term "short" esophagus.

I would like to emphasize that I believe many people are denied the proper surgical correction of esophageal hiatus hernia, frequently because the radiologist tells the referring doctor that the lesion is a "short" esophagus. Since short esophagus has carried with it a very poor repair history, the careless, repetitious use of the word in x-ray reports and in ordinary consultation room chatter means that too many patients who should have their hiatus hernia repaired are not offered the needed surgery.

In the literature, even within the past few months, x-ray reproductions are shown which are entirely similar to those presented by the authors, namely, the round, ball-like projection of the stomach into the chest.

Many of these are called short esophagus, but I have yet to operate upon one of these patients and be unable to pull the esophagogastric junction down beneath the diaphragm where it belongs. It takes a certain amount of gentle pulling, but such a stomach can be reduced with perfect impunity.

Therefore I submit that we should get away from saying anyone has a short esophagus, when indeed the esophagus is not short. It can be lengthened perfectly well, as we have repeatedly proved to our own satisfaction.

I would like to describe another means by which one can always tell whether or not the esophagus can be lengthened. If at esophagoscopy the esophageal gastric junction can be moved 2 or 3 cm., one is absolutely certain that in surgery this particular junction can be pulled down below the diaphragm.

I enjoyed discussing this presentation very much and apologize for going a little away from the exact subject of the paper, but it is a very important excursion to my mind.

I think we should all be aware of the words we are using when we talk about short esophagus.

ORVILLE F. GRIMES (closing): I appreciate the remarks of those who have discussed this paper. The discussion has added much to the understanding of the problems associated with the surgical treatment of esophageal hiatus hernia.

During this study it became apparent that the results of the surgical treatment of esophageal hiatus hernia were rather good, regardless of the type of repair accomplished. This was true whether the operative repair was done transthoracically or through an abdominal approach. The same could be said about good results whether the phrenoesophageal ligament was sutured to the undersurface of the diaphragm or whether the hernia was merely reduced from above, with closure of the diaphragmatic crura thereafter.

These good results can be interpreted to indicate that if the successful replacement of the cardioesophageal junction below the level of the diaphragm is accomplished, two of the three sphincteric mechanisms discussed in this presentation will have been restored: the cardioesophageal angle is replaced and the diaphragmatic muscular sling is again made more effective. If in addition the patient maintains a functioning inferior esophageal constrictor, all three of the anatomic and physiologic sphincter mechanisms are restored. Lastly, if the phrenoesophageal ligament is shortened and sutured below the level of the diaphragm, it again may become an antagonist to the longitudinal muscle pull of the esophagus. This latter procedure would then restore completely the anatomy and physiology of the normal esophagus and the esophageal orifice of the diaphragm.

Right Phrenohepatic Incarceration

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INCARCERATION of the liver through a traumatic defect in the right diaphragm occurs rarely, but represents a specific disorder. The rare incidence of right-sided diaphragmatic hernia due to trauma has been attributed to the protection afforded the diaphragm by the liver. It has been stated that unless the laceration of the diaphragm was large enough to allow herniation of the entire lobe of the liver, such displacement was unlikely to occur [*1*]. This is not always the case, however, and it is the purpose of this paper to focus attention upon a pathologic entity which we have termed right phrenohepatic incarceration to distinguish it from other types of diaphragmatic herniation.

During the past few years we have treated eight such cases. The first four were reported in 1955 [*2*]. In view of the knowledge gained from the management of the additional cases, they will be described in this report. A review of the entire series emphasizes the consistent characteristics of the disorder.

The pathologic process consists of protrusion and incarceration of a portion of hepatic parenchyma through a defect in the right diaphragm following injury. There appears to be progressive herniation of liver substance with gradual molding of the hernia mass which assumes a characteristic mushroom-like form in many cases. A history of severe injury, usually associated with an automobile accident, has been obtained in each case. We believe that the increased incidence evident in our own experience reflects the increasing number of traffic accidents in general.

Traumatic hernia of the diaphragm is not unusual following injury to the chest or abdomen. It is well established that herniation of abdominal viscera is much less apt to occur through the right leaf of the diaphragm than through the left [*3*]. Hedblom found the relative frequency to be 5 per cent on the right and 95 per cent on the left with fourteen instances of liver herniation in 857 cases of diaphragmatic hernia [*4,5*]. In a review of fifty-seven cases, Evans and Simpson found severe diaphragmatic hernias due to non-penetrating injuries, one of which occurred on the right [*6*]. Other authors have reported isolated cases of liver herniation, some which simulated intrathoracic tumors and others with strangulation of the herniated liver [*1–10*]. It is possible that reported accessory lobes of the liver [*11,12*] extending through the diaphragm into the thorax may be the result of previous trauma. Certain aspects of phrenohepatic incarceration differentiate it from herniation of the more mobile viscera through the diaphragm [*13*], the demonstration of which is emphasized in the following case histories.

CASE REPORTS

CASE 1. A. M., a sixty-six year old woman, was seen because of fever, pain in the right chest and right shoulder, and the finding of a right supradiaphragmatic shadow on chest roentgenogram. Her symptoms were of two weeks' duration, were spontaneous in nature and were not precipitated by any recent injury. The patient gave a history of being in a severe automobile accident ten years previously in which her husband was killed. She was pinned beneath the car with considerable weight over her right abdomen for a period of thirty minutes and was hospitalized for several days and discharged without evidence of serious trauma. Chest x-rays immediately after the accident showed no abnormality. Following discharge from the hospital, she had no symptoms until those which brought about her present hospital admission.

Physical examination revealed the patient to be markedly obese, weighing 280 pounds. Other than her obesity, abnormal findings consisted of leukocytosis of 18,000 with pain in the right chest and right shoulder. Roentgenograms and fluoroscopy of the chest showed a spherical 5 cm. mass at the right base. (Fig. 1.) The mass was in close approximation to the diaphragm. The right costophrenic gutter was blunted and there was slight thickening

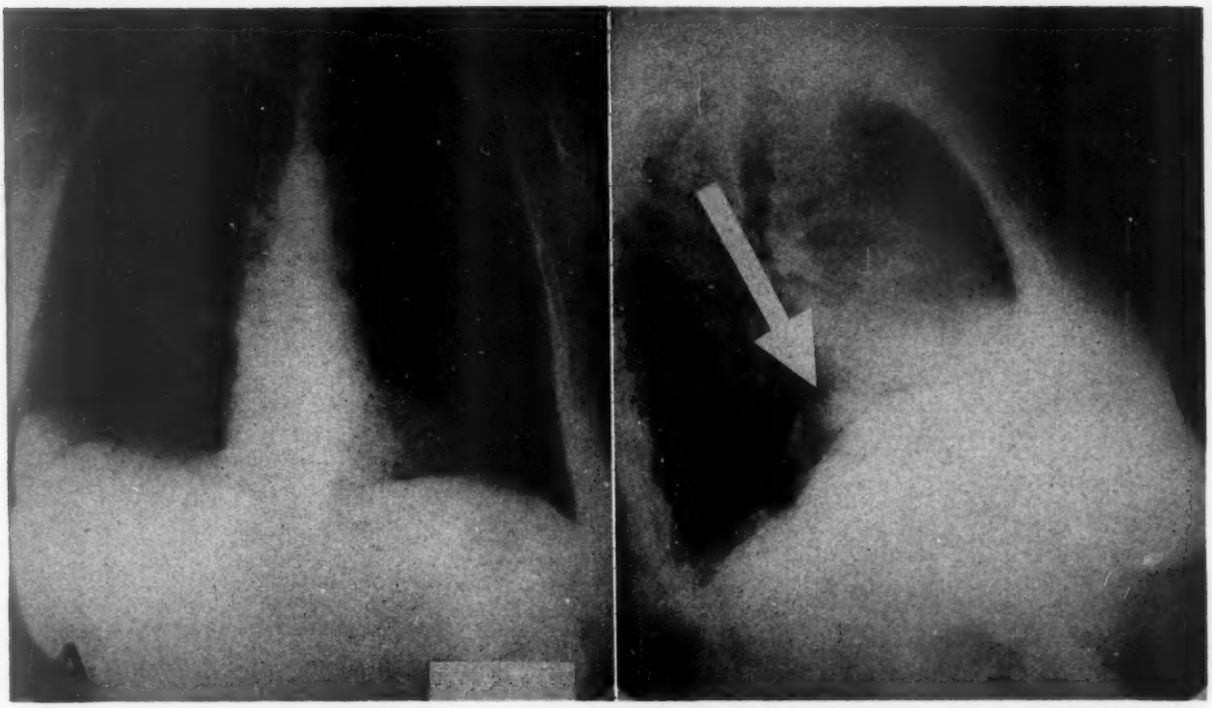

FIG. 1. Case 1. A, chest roentgenogram showing spherical mass at right base representing incarcerated liver. B, lateral projection demonstrating mid-diaphragmatic location of herniated liver.

of the minor interlobar septum. Elsewhere the lung fields were clear. The heart was normal in size, shape and position. The impression from a roentgen standpoint was probable traumatic hernia of the right diaphragm with incarceration of the liver.

Thoracotomy was performed on December 15, 1954, and because of the patient's tremendous size, it was decided to confine the approach to an anterior incision, which was made through the fifth interspace. Following insertion of the rib spreaders, it was noted that the pleural cavity contained approximately 75 cc. of a musty, slightly cloudy fluid. The lung was densely adherent to the upper surface of a mass directly over the diaphragm and these adhesions were separated by sharp dissection. There was no sac present and the liver parenchyma was easily recognized as a mushroom-like mass as soon as the lung was freed. After freeing the pedicle and enlarging the opening in the diaphragm, the liver was readily reduced. The diaphragm was closed with interrupted mattress sutures. The patient had an uneventful postoperative course and was discharged ten days following surgery.

Comment. It is apparent from this patient's history that unless the initial trauma is of such a nature as to demand immediate surgical exploration, injury to the diaphragm may be unsuspected and pass unnoticed. A latent interval of an undeterminate period of time may intervene between the accident and the onset of symptoms. This period may vary from a few months to many years.

CASE II. R. F., a fifty-four year old man, was referred for thoracic consultation because of repeated respiratory infections and progressive changes on chest x-ray. He had been under close medical supervision for several years because of x-ray findings of a paralyzed right diaphragm, but his symptoms were remarkably few. When first seen by us on September 10, 1954, chest roentgenograms showed a mass occupying the lower half of the hemithorax with no definite right diaphragmatic shadow. The left lung was clear. Fortunately, previous x-rays were available and comparison of chest films taken at regular intervals since 1949 showed progression of the lesion on the right side. (Fig. 2.) Careful history elicited a severe automobile accident occurring twelve years before in which the patient was jarred by a head-on collision. He had several fractured ribs on the right, but no other thoracic injury was noted on the chest x-rays and he was not hospitalized.

At the present admission, physical examination revealed a somewhat pale, middle-aged man of better than average body build. He was 6 feet, 2 inches tall and weighed 250 pounds. The patient had been employed as a mail carrier for the past twenty-five years and had been able to work with-

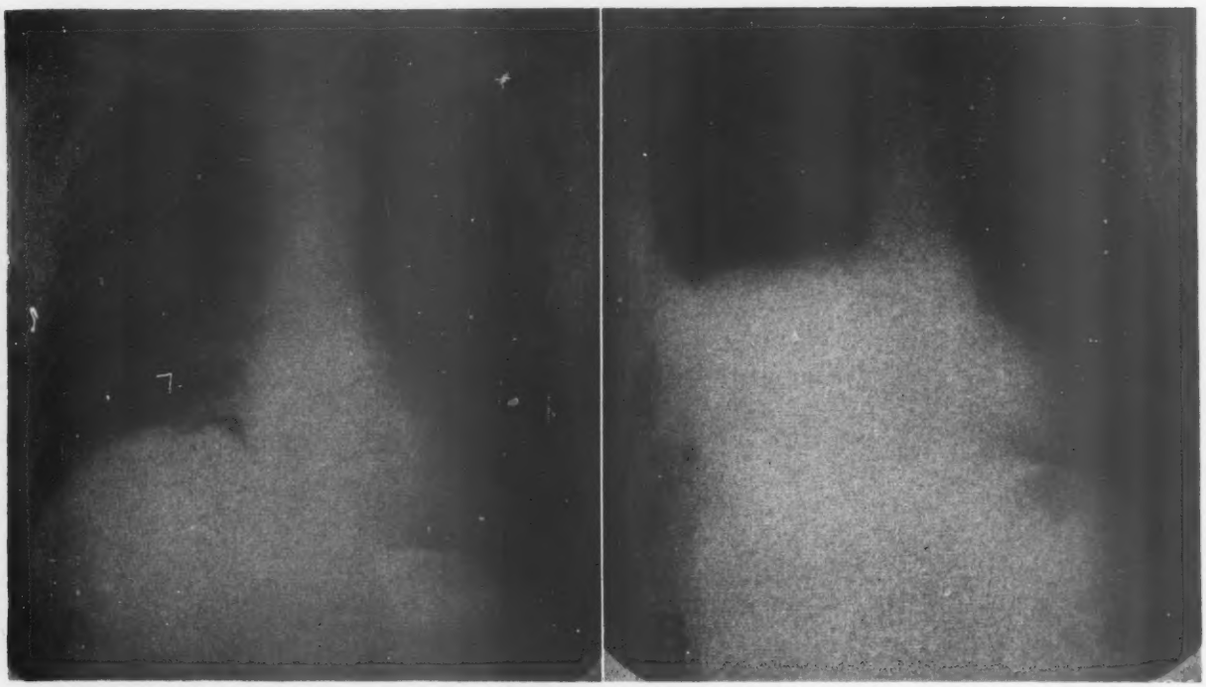

FIG. 2. Case II. A, chest roentgenogram taken in March, 1949, seven years after severe injury to body. Note elevated right diaphragm. B, x-ray of chest five years later showing further involvement of right diaphragm due to progressive liver incarceration.

our interruption except for brief absences due to respiratory infections. While the impression of right diaphragmatic hernia with liver incarceration was considered, bronchoscopy was performed during the course of his clinical work-up. This showed only a narrowing of the middle lobe bronchus as if from extrinsic pressure. Right thoracentesis was productive of 20 cc. of uncharacteristic clear fluid. A leukocytosis of 15,000 completed the abnormal findings. A diagnosis of right phrenohepatic incarceration was made and thoracotomy was performed on September 23, 1954. With the patient in the left posterolateral position, the chest was opened through the bed of the resected sixth rib. Upon entering the pleura, it was apparent that there was a large amount of abdominal content within the thoracic cavity. The entire liver presented itself in the chest and the gallbladder was rotated and presented immediately beneath the pleura. A large segment of the omentum was present as well as a portion of the transverse colon. At this point, the patient's blood pressure became unobtainable and the anesthesiologist inquired as to the action of the heart. The heart action was slow, but regular and it was believed that by lifting the liver mass away from the region of the inferior vena cava, the patient's condition might improve. Following this manipulation, the vital signs immediately returned to normal. The diaphragmatic opening was exposed and stay sutures applied to its edges. The defect was located anteriorly in the right portion of the diaphragm and midway between the mediastinum and the lateral chest wall. It was necessary to enlarge this opening in order to return the liver into the peritoneal cavity. This was accomplished with considerable difficulty because of the large mass that had to be replaced. The thickened edges of the hernia were then easily approximated. The lung expanded under positive pressure satisfactorily and the chest was closed. The patient made a satisfactory postoperative recovery and was discharged from the hospital in nine days.

Comment. During the five-year period that this man was under medical observation, there was progression of the lesion. The roentgenographic findings in 1949 were interpreted as a paralyzed right diaphragm which was unexplained. The mass appeared gradually. It is unusual for severe physiologic disturbances to occur in the right-sided type of herniation involving liver alone. Disturbances of gastrointestinal or cardiopulmonary physiology frequently occur with hernias on the left. In spite of a herniated colon and gallbladder, this patient had had no symptoms referable to these organs which might be attributed to the hernia, nor had he had other vascular symptoms prior to surgery. He experienced severe circulatory

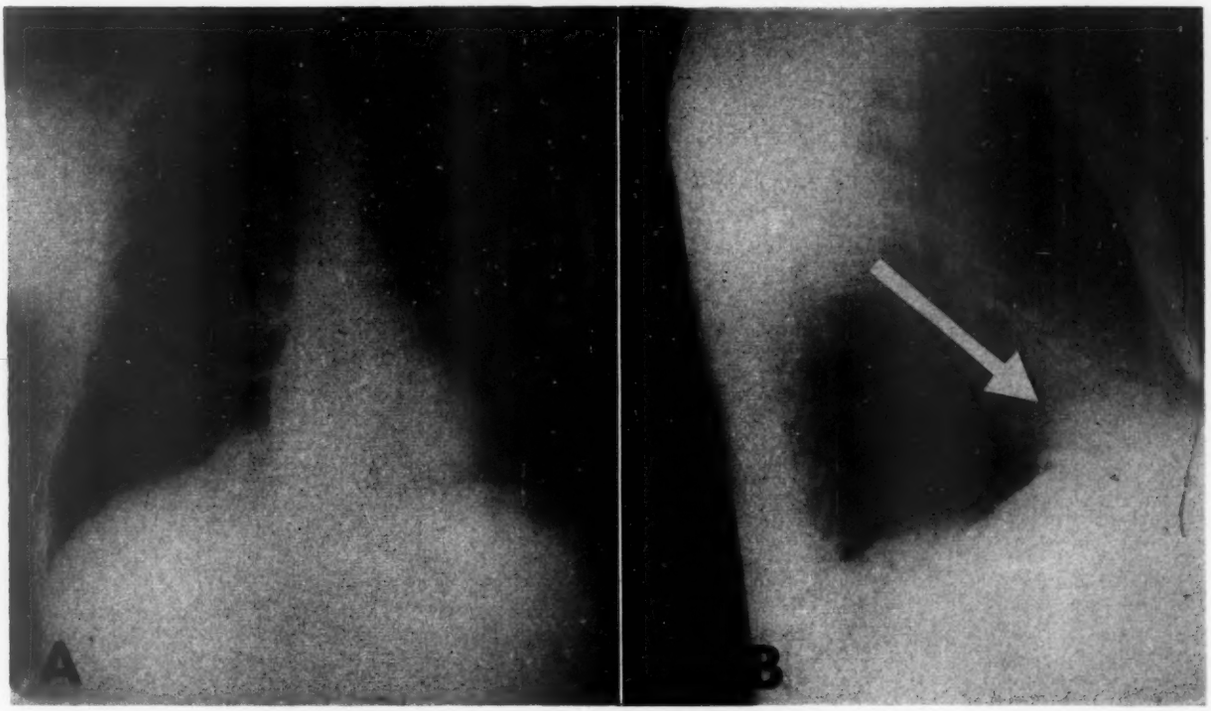

FIG. 3. Case III. A, right cardiophrenic mass in asymptomatic patient. B, lateral view showing anterior location of incarcerated liver mass.

embarrassment when the involved thorax was opened. Compression of the vena cava by the herniated liver was completely relieved by the reparative procedures.

CASE III. F. M., a forty-three year old man, was seen on October 10, 1956, having been referred because of an undiagnosed right cardiophrenic mass. (Fig. 3.) The lesion was discovered during the course of a routine physical examination including chest roentgenograms. His past history was uneventful with the exception that he reported a very serious automobile accident which occurred in the spring of 1937, at which time he had a skull fracture, his lung was collapsed and he was unconscious for sixteen days. At the time of the accident, he was thrown from the automobile for a distance of ten or fifteen feet. He recovered satisfactorily from this incident and the chest x-ray at the time of the accident showed no abnormality.

Physical examination revealed a man of rather short stature (5 feet, 10 inches) weighing 195 pounds, with no essential abnormality outside of the lesion in the right chest noted on x-ray. He had no pain or discomfort in the chest area and his only concern was the possibility of a neoplastic lesion in the right chest. Prior to his referral, bronchoscopy and bronchography were performed but showed no evidence of abnormality. Likewise, pneumoperitoneum indicated that there was no definite communication between the diaphragm and the right pleural cavity. Laboratory studies were within normal limits except for mild leukocytosis. Because of the unknown nature of this lesion, the patient was admitted to the hospital where right thoracotomy was performed through the right sixth anterior interspace. A mass immediately presented itself above the anterior leaf of the diaphragm. The lung was not adherent to the mass which was identified as liver. With retraction of the lower and middle lobes, it was noted that there was a small amount of clear, free fluid in the pleural cavity. It was apparent that there was early mushrooming of the liver over the upper surface of the diaphragm. There was definite strangulation of the liver substance through the diaphragmatic opening and a pleural sac over the liver prevented communication between the thorax and the abdomen. An opening was made in the pleural ring at the base of the incarcerated liver which was then returned to the abdominal cavity without necessitating any enlargement of the diaphragmatic opening. The firm edges of the diaphragm were readily closed and the lung was expanded satisfactorily. Following closure of the chest, the patient made an uneventful recovery and was discharged in ten days.

Comment. This case raises three important points of interest. First, the accident in which

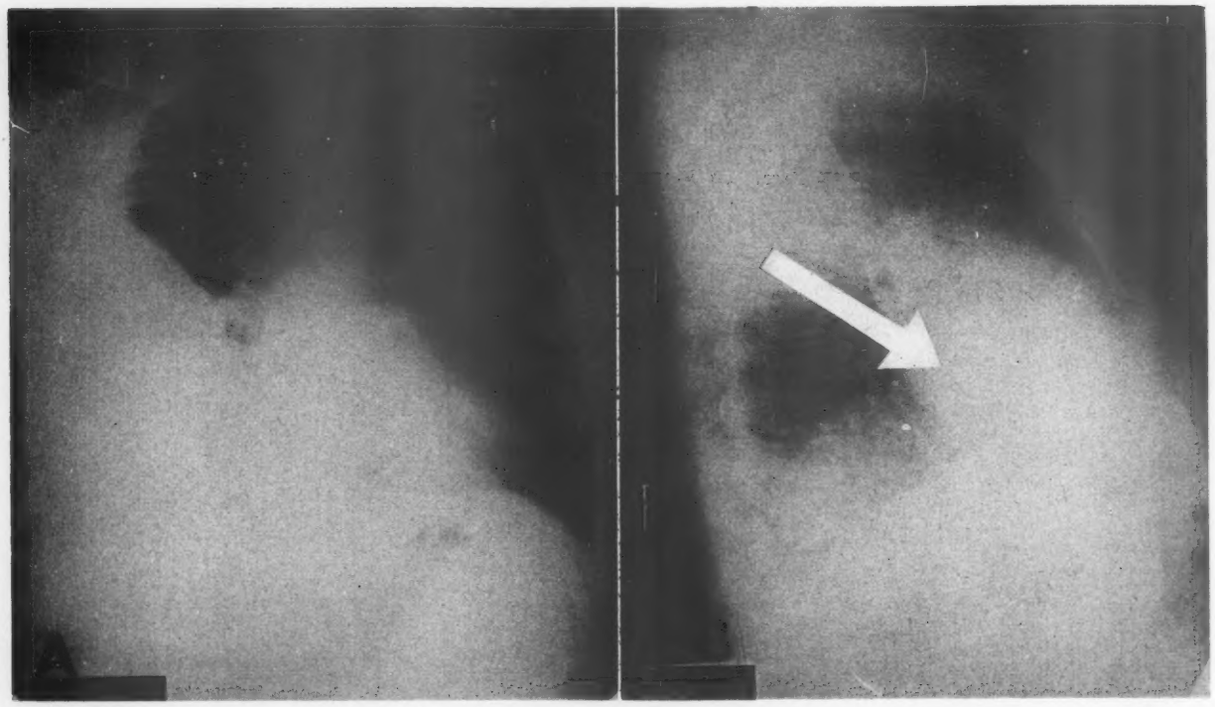

FIG. 4. Case IV. A, recurrent right diaphragmatic hernia with liver incarceration five years following original repair. B, lateral chest film showing anterior location of hernia mass.

the patient was involved was very severe. Massive contusion of the trunk with compression of the abdomen associated with accidents of this type, we believe, has etiologic significance. Second, thoracotomy was necessary to make a diagnosis. The possibility of a neoplastic lesion in the right chest had been strongly considered. Third, the herniated liver showed evidence of strangulation.

CASE IV. E. V., a forty-four year old man, was seen in December, 1955. In April, 1950, the patient sustained a stab wound of his chest, a steel bar having been driven through the right anterior chest at the level of the fifth rib and penetrating the right posterior chest wall. He was hospitalized and was maintained on transfusions and supportive measures for forty days, at which time repair of a traumatic hernia of the right diaphragm was performed. Subsequent to the operative procedure, the patient did well except for a peculiar psychotic episode which confined him to the hospital for three months. It is of interest that during this hospitalization he gained 50 pounds, which weight he maintained. Following this discharge, he did well until November, 1955, at which time increasing symptoms of pain and spasm developed in the right lateral chest at the point of exit of the steel bar. Because of these complaints, the patient was referred for further evaluation and possible therapy. The past history was essentially negative except for a traumatic amputation of the left arm several years prior to this admission.

The patient was a man of short stature (5 feet, 9 inches) and weighed 250 pounds. Examination of the chest revealed a soft bulge at the site of the point of exit of the steel bar which became more pronounced with coughing. The lung showed a marked decrease in breath sounds over the right anterior and lateral chest. There was some wheezing in the right chest posteriorly at the level of the fifth interspace. The heart was normal and the remainder of the physical examination was noncontributory. Roentgen and fluoroscopic examination of the chest revealed a large opacity anterior in the lower right hemithorax which extended approximately to the level of the third rib anteriorly and moved paradoxically with inspiration. (Fig. 4.) The right leaf of the diaphragm was indiscernible from the mass. Barium swallow revealed no abnormalities of the esophagus and a stomach normal in outline and position. No hiatus hernia was demonstrated and the course of the small bowel did not appear unusual.

The patient was taken to the operating room on January 16, 1956, where right thoracotomy was performed through the fourth interspace. Dense adhesions were encountered upon entering the chest, and dissection was performed carefully and the right lower lobe freed. In dissecting the lower lobe, a large mass was encountered which protruded

through an opening in the right diaphragm. Further dissection of this area revealed omentum in the right pleural cavity together with a large cup-sized portion of the liver which had likewise herniated through the opening in the diaphragm. Careful dissection was necessary to free the omentum and the strangulated portion of the right dome of the liver from the hernial orifice, and these structures were then reduced into the peritoneal cavity. The diaphragm was closed with interrupted sutures, and the lung was expanded in the usual manner. Postoperatively, the patient did well. A chest tube was removed on the day following surgery and he was released on the eleventh postoperative day in good condition.

Comment. While this patient had a definite incarcerated liver, the exact etiology differs from the other cases. Although an adequate repair of the original hernia was undoubtedly done, we believe the tremendous weight gain caused undue pressure on the already weakened right diaphragm with the development of an incarcerated hepatic hernia.

A review of our entire series of eight patients shows the constant findings in right phrenohepatic incarceration depicted in Table 1.

COMMENTS

The clinical characteristics of incarceration of a portion of the liver through a traumatic right diaphragmatic hernia are well demonstrated by these cases. There is a history of injury, often incurred in an automobile accident and usually of such severity as to require hospitalization. Symptoms are delayed and may be obscure or relatively absent. These consist of pain in the chest and upper abdomen, usually on the right and variable in nature. Occasionally, the pain may be disabling. It is remarkable that all of these patients have been of a heavy body build. We believe that obesity is a factor in the pathogenesis of this disorder as it may be in other types of diaphragmatic hernia. The roentgen characteristics are interesting. The lesion usually presents as a radiographic finding of a mass in the right chest just above and intimately associated with the diaphragm. There may be a characteristic mushroom-like appearance to the mass, depending upon the size of the diaphragmatic defect and the extent of the herniation. Moderate leukocytosis has been present in each case ranging from 18,000 to 22,000.

The condition is treated surgically by reduction of the liver and repair of the diaphragm through right thoracotomy. In our cases we have found no indication for resection or removal of the herniated portion of the liver as has been previously reported [8]. Indeed, although loss of the right of domicile may occur with very large herniations, as in the second case, reduction is usually not difficult. The operative findings have been presented. Surgical correction is indicated because of liver strangulation due to pressure of the diaphragmatic rim, pain in the chest and occasionally, for the invalidating aspects of symptoms. Repair in each case has been curative with no recurrence or mortality.

TABLE 1
CONSTANT FINDINGS IN RIGHT PHRENOHEPATIC INCARCERATION

Clinical:	
1.	Past history of severe injury
2.	Obesity
3.	Roentgen appearance of a right diaphragmatic lesion
4.	Leukocytosis
Operative:	
1.	Incarcerated liver
2.	Pleural fluid
3.	Anterior diaphragmatic defect with firm edges

Other disorders which may present as a mass in this region may be indistinguishable from incarcerated liver, and a definite diagnosis must be made by thoracotomy. The differential diagnosis must include primary diaphragmatic tumors, congenital lesions of the diaphragm, segmental diaphragmatic paresis secondary to phrenic nerve involvement and tumors of the lung and mediastinum. Loculated pleural effusions, omental hernias through Morgagni's foramen, pericardial cysts, or cardiophrenic fat pads also may have a similar radiographic appearance. One must also consider pathologic processes confined to the liver, but causing distortion of the diaphragm.

Induced pneumoperitoneum and pneumothorax are specialized radiographic technics which may be used on occasion to aid in the differential diagnosis of lesions of this type. With pneumoperitoneum, one may or may not be able to distinguish between a subdiaphragmatic lesion and incarcerated liver. In this connection, the associated inflammatory changes which occur about the hernia ring and the

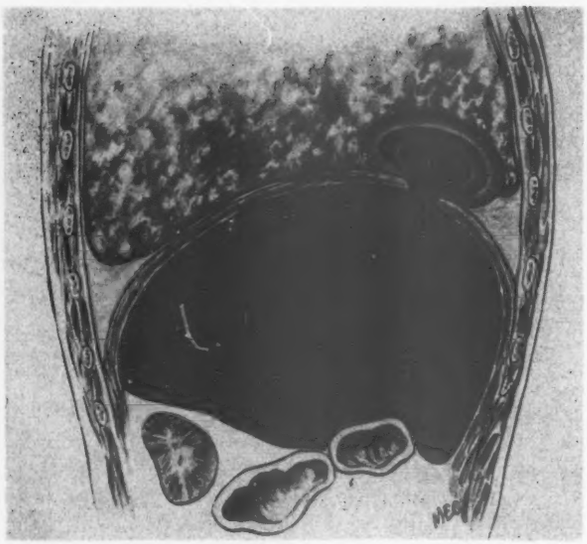

FIG. 5. Sagittal sketch showing progressive liver incarceration.

extreme tightness with which the liver herniates may prevent air from entering the pleural cavity. Negative findings with pneumoperitoneum do not rule out the possibility of this lesion. Pneumothorax may distinguish a primary lesion of the lung as it falls away from the diaphragm after introduction of the air. A definite diagnosis depends upon the clinical findings and the identification of a lesion by thoracotomy.

The pathogenesis of this type of diaphragmatic hernia poses several interesting possibilities. The delay between the time of injury and the onset of symptoms together with the gross characteristics of this lesion suggest its gradual development. It is our impression that incarceration of a portion of the liver as seen at surgery does not occur immediately at the time of injury. Rather, an initial local weakening or split in the diaphragmatic muscle occurs at its most superior portion—anatomically, this is the anteromedial segment of the right diaphragm. With the passage of time, there is a molding of the liver parenchyma through this splayed diaphragmatic muscle with the resultant mushrooming and eventual hepatic incarceration. (Fig. 5.) In several of these cases, a hernia sac was found. This might be considered unique, since the absence of a sac has been considered one of the cardinal features of traumatic diaphragmatic hernia. We believe, however, that in these instances the original injury of the diaphragm consisted of a splitting of the muscular portion with preservation of the integrity of the diaphragmatic pleura. This, in time, becomes the hernia sac.

It has been noted that the injury was evident in eight patients, all of whom were of a heavy body build. It is suggested that obesity predisposes the patient to this type of injury because of increased intra-abdominal pressure. Over a period of time the combined effect of positive intra-abdominal and negative intrathoracic pressures result in the progressive molding of the liver parenchyma through the rent. It may be recalled that esophageal hiatus hernia is most common in obese patients. The aggravating effect of positive abdominal and negative thoracic pressures has been discussed by Allison [*14*] and Madden [*15*], with reference to this type of hernia. The presence of the lung may account for the flattening of the protruded liver which gives the characteristic mushroom-like appearance. A flattening and descent of the diaphragm which accompanies aging and emphysema may also be a factor. Free fluid is often found in the involved pleural cavity. These findings suggest the strangulating effect of a hernia ring upon its contents.

SUMMARY

Four cases of traumatic hernia of the liver through the right diaphragm are added to the previously reported cases. The descriptive term, right phrenohepatic incarceration, has been suggested. The common clinical characteristics of this abnormality are given. The probable pathogenesis of the disorder is discussed.

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DISCUSSION

ORVILLE F. GRIMES (San Francisco, Calif.): Drs. Dugan and Merrill have called to our attention an interesting clinical entity. One usually visualizes the liver as a solid unyielding structure whose configurations and attachments are unchanged except by processes indigenous to the hepatic parenchyma itself. The topographic changes of the liver incident to tumors, areas of regeneration and asymmetric enlargement due to hepatitis are commonly in mind when an x-ray of the chest demonstrates an abnormal shadow in the right diaphragmatic area. The authors add a new concept which suggests that the liver has the capacity to mold itself, piecemeal so to speak, into false body cavities in a manner somewhat similar to the migration of softer, more resilient and elastic organs.

The authors' concept of gradual development of the liver herniation through a weakened area in the right diaphragm due to trauma is valid and reasonable. The delay between recognizable symptoms and the known injury lends support to the slow but relentless development of the entity. It seems justifiable, however, to assume that two other possible mechanisms may be involved in the formation of such a hernia. First, one can visualize an acute herniation of the liver through a sudden rupture of the right diaphragm with incarceration occurring immediately and maintained by the differential positive and negative pressure relationships in the abdomen and thorax, respectively. In such a circumstance the late development of symptoms could be explained by the gradual tightening by fibrosis of the diaphragmatic ring about the imprisoned liver. Second, it can be reasoned that the same force which causes weakening of the diaphragm also may result in local hemorrhage, perhaps contained in a localized area by virtue of the attachments of the liver to the diaphragm. In the resolution of such a hemorrhage, further weakening of the diaphragm may occur either from the proteolytic effect or from the pressure generated by the accumulating fluid within the localized hematoma. This would serve perhaps to make an otherwise tenuous but still intact diaphragm a deficient one in the local area of trauma.

Our experience with this problem at the University of California Hospital has been extremely limited. One interesting problem can be recorded, however, which will serve to emphasize the apparent ease by which the diaphragm can be ruptured by any one or a combination of these factors. A fifty-two year old white man underwent right pneumonectomy for carcinoma of the right main stem bronchus in April, 1950. The right phrenic nerve was deliberately sectioned. Two years later the patient complained of pain in the right upper anterior chest, noted especially when he was constipated. Coupled with this was the fact that a persistent air fluid level had been noted in the apex of the right chest without symptoms to suggest that such was due to empyema or bronchopleural fistula. A barium enema showed that the hepatic flexure of the colon lay in the apex of the right chest. The most plausible explanation is that the pleural fluid in the right chest with its attendant reaction postoperatively added a weakening effect to an already paralyzed diaphragm. Along with this, perhaps some differential pressure relationships existed in this obese patient so that the additive effect of these several factors was sufficient to cause a delayed diaphragmatic rupture.

JOHN F. HIGGINSON (Portland, Ore.): I have had an opportunity to see only two such cases, but because the authors said that trauma was unquestioned in etiology, I thought I should mention the fact that one of my cases definitely was traumatic in origin and one apparently was not.

Neither patient was fat; one was a man, the other a woman. The man had mild emphysema. The woman had a right, which she exercised, to be proud of her figure.

Both patients had plugs of liver, which looked like doorknobs, in the type of deformity that the authors demonstrated in Figure 5, but they were in the posterior half of the right diaphragm.

The woman had been in an automobile accident, and I think typifies the group of cases presented by the authors.

The man had had a negative x-ray a number of years before the x-ray was taken which brought him to my attention.

The reason for performing exploration upon these patients was that no diagnosis was made, and it was suspected that there might be a tumor, as has been outlined by the authors.

ROSS ROBERTSON (Vancouver, B. C., Canada): I have a case to add to the authors' series.

This was a forty-five year old woman with no history of trauma, and a long series of gastrointestinal complaints, recurrent attacks of nausea and vomiting, and abdominal pain, usually coming on after lifting. Intensive investigation including laparotomy revealed no abnormalities. Ordinary chest x-ray was negative, until one radiologist took a lateral x-ray which revealed a rounded herniation of liver through the right diaphragm.

At operation the diaphragm had a hole in it about 2 inches in diameter. Bare liver tissue herniated through this hole, mushrooming above to a diameter of 3 inches. A finger could be inserted down beside the liver into the free peritoneal cavity.

It seems possible that during her attacks there was herniation of either small bowel or omentum which caused her symptoms.

DUANE L. MERRILL (closing): We have been intrigued by the progressive pattern taken by right phrenohepatic incarceration, and would like to cite a case which further demonstrates, we believe, that an injury to the diaphragm is not a static process.

However, first I would like to suggest that possibly some of the liver protrusions in the posterior portion of the diaphragm may represent congenital defects of one type or another. A history of injury has been found in each of our cases, but it is entirely possible that segmental diaphragmatic lesions can occur from other causes, and that the progression of the liver through the diaphragm might continue from that point on.

In order to prevent overspecialization, I am going to talk about the left side of the diaphragm.

This patient had a traumatic hernia through the left side of the diaphragm. Initially he presented with elevation of the left diaphragmatic leaf. His complaints were that he was unable to swallow and had had some symptoms of hematemesis for a period of two months.

A barium swallow and an upper gastrointestinal series revealed that the stomach was involved in an apparent hernia into the left thorax with almost complete volvulus of the stomach; there was no passage of barium beyond the pylorus.

We utilized pneumoperitoneum in studying this case and it was quite eventful, in that following the pneumoperitoneum the stomach was completely reduced from a fairly large hernia sac. A repeat barium swallow showed that the barium then continued on through the duodenum into the small bowel.

Subsequently the patient was operated upon, and the sac was visualized through thoracotomy.

This was an interesting hernia, and I chose the case as an example for the following reasons: There were fractures of the ninth and tenth ribs. Adjacent to this the diaphragm was split and a nubbin of spleen protruded into the thorax. The laceration continued a little more medially. Lying next to this there was a small amount of adherent omentum, and from that point on the diaphragm was not completely torn, the diaphragmatic pleura being intact and forming the large sac. It was within this sac that the stomach was incarcerated.

The laceration continued medially as a disruption of all of the layers of the diaphragm, except the diaphragmatic pleura which formed the large sac seen on x-ray.

This case is cited merely to substantiate the pathogenesis of right phrenohepatic incarceration, as presented.

The development of a sac such as this, similar in many respects to sacs found in some of the cases of liver herniation which have been reported, is obviously a delayed process.

Some patients do not have sacs. We believe that in these instances the initial tear in the diaphragm is complete and that the herniation continues, as demonstrated in the second case.

The Surgical Removal of Metastatic Malignant Disease of the Thyroid Gland from the Anterior-Superior Mediastinum

H. J. McCorkle, M.D., *San Francisco, California*

From the Surgical Research Laboratories of the University of California School of Medicine, San Francisco, California.

It has been observed at operations and autopsies performed on patients with cancer of the thyroid gland that the disease may extend or metastasize to the anterior-superior mediastinum. In 1952 a method for dissection of the anterior mediastinum, devised in the anatomy laboratory and applied to the treatment of four patients with operable cancer of the thyroid gland was reported [1]. Mediastinotomy was performed in two other instances in which the tumor was found to be inoperable and the malignant disease was only partially removed. Other surgeons have reported experiences with a similar procedure [2–5].

Further experience with superior mediastinal dissections has resulted in some alteration and extension of the procedure. The anatomic and surgical dissections are similar and, therefore, are described together. A straight incision is made over the sternum from the center of the usual transverse thyroidectomy incision. (Fig. 1.) The sternum is incised from the sternal notch to the fifth intercostal space and spread with retractors. Great care is required to dissect the pleura from other mediastinal structures and to preserve it. In some cases a part of the pleura is removed if it is adjacent to diseased tissue, and the resulting pleural defect is repaired or grafted with fascia. Removal of the anterior-superior mediastinal tissues begins with dissection of the areolar tissue from the pericardium, and the dissection extends upward to remove a segment of tissue containing the mediastinal areolar tissue, lymphatics and thymus in continuity with the similar tissues in the lower anterior part of the neck. (Fig. 2.) The phrenic nerves are the lateral boundaries of the dissection, and these nerves as well as the vagus and recurrent laryngeal nerves are clearly defined and preserved. The great vessels including aorta, superior vena cava, innominates, right subclavian and carotids are carefully stripped of areolar tissue and lymphatics in a manner similar to that used in cleaning the axillary vessels in radical mastectomy. (Fig. 3.) A part of the thoracic duct has been removed in some dissections. It should be emphasized that these surgical procedures should be performed where facilities are available for intubation for intratracheal anesthesia, as is usual for intrathoracic procedures, and by surgeons familiar with thyroid and thoracic surgical procedure. The mediastinal dissection is somewhat more easily performed at the time of total thyroidectomy, but it can be done at a later stage as a second operation. The pleura is carefully examined and tested for airtightness before closure of the incision is begun. The sternum is repaired with stainless steel wires. Drainage is provided through small catheters to which continuous aspiration is applied. The dressing should be airtight.

So far this procedure has been applied only to patients with cancer of the thyroid gland with evidence of metastatic spread toward the mediastinum, usually with metastases in the lymph nodes at the lower border of the thyroid isthmus, inferior portion of a thyroid lobe, or

FIG. 1. Illustration indicating surgical approach used in mediastinal dissection for carcinoma of the thyroid gland. (From: McCORKLE, H. J. *West. J. Surg.*, 63: 324–333, 1955.)

with palpable or x-ray evidence of intrathoracic spread.

Neck dissection has been performed at the same time as thyroidectomy and mediastinal dissection, but it makes the procedure very long and it probably is preferable to do neck dissections at later stages. This is particularly so in slow-growing types of thyroid neoplasms, for the neck can readily be examined at follow-up visits whereas the mediastinum cannot.

In two of the three patients in whom neck dissection was combined with thyroidectomy and mediastinal dissection, tension pneumothorax developed. These two patients also had temporary parathyroid deficiency. Both have recovered from their complications. In all other cases the postoperative course was not particularly difficult and produced no disability.

Nine more patients have been treated in addition to the six cases previously reported. The records of these fifteen patients in whom mediastinal dissection has been performed for cancer of the thyroid gland are summarized in Table 1.

SUMMARY

1. Studies made at autopsy and at surgery in patients with cancer of the thyroid gland indicate that the disease may spread to lymph nodes below the thyroid isthmus and into the anterior-superior mediastinum.

2. A method for dissection of the anterior-

TABLE 1

SURGICAL REMOVAL OF THE SUPERIOR MEDIASTINAL LYMPHATICS FOR CANCER OF THE THYROID GLAND: SUMMARY OF CASES

Patient, Sex and Age (yr.)	Type of Growth	Location		Procedure	Follow-up
		Primary	Involved Nodes		
A. S., F, 51	Adenocarcinoma	Isthmus	Below isthmus in left inferior region of thyroid	Total thyroidectomy; mediastinal dissection	No recurrence 3½ yr. postoperatively
R. C., M, 60	Undifferentiated cellular pathology, nodular	Left lobe	Left substernal; mediastinal; bilateral cervical	Total thyroidectomy; mediastinal dissection; bilateral neck dissection	No evidence of recurrence 3½ yr. postoperatively
C. P., F, 40	Undifferentiated cellular pathology	Undetermined (entire gland involved)	Superior mediastinal mass surrounding and involving innominate vein and left carotid artery	Surgical exploration of neck and mediastinum; tracheotomy; palliative partial excision of tumor	Died 1¼ yr. postoperatively
O. V., M, 62	Mixed papillary and undifferentiated carcinoma	Right lobe	Anterior-superior mediastinal, involving trachea and innominate veins	Bulk of tumor removed; tracheotomy	Died 1 mo. postoperatively
M. K., F, 22	Papillary adenocarcinoma	Left lobe	Below and to left of isthmus; left cervical	Total thyroidectomy; left radical neck dissection; mediastinal dissection	Firm node in submaxillary triangle 4 yr. postoperatively
J. C., F, 27	Papillary carcinoma	Left lobe	Left cervical; mediastinal	Total thyroidectomy; left radical neck dissection; mediastinal dissection	No recurrence 4 yr. postoperatively
M. M., F, 57	Adenocarcinoma	Isthmus	None	Total thyroidectomy; mediastinal dissection	No recurrence 3 yr. postoperatively
E. K., F, 34	Papillary adenocarcinoma	Left lobe	Mediastinal and bilateral cervical	Total thyroidectomy; mediastinal dissection; bilateral neck dissection	No recurrence 3¼ yr. postoperatively
R. B., M, 22	Mixed papillary and poorly differentiated	Isthmus	Bilateral cervical and mediastinal	Total thyroidectomy; bilateral neck dissection; mediastinal dissection	No recurrence 4 yr. postoperatively
G. S., F, 50	Papillary	Diffuse	Mediastinal	Mediastinal dissection (thyroidectomy had been performed 4 yr. earlier)	No evidence of recurrence 3 yr. postoperatively
R. J., M, 30	Papillary adenocarcinoma	Right lobe	Bilateral cervical; suprasternal notch; mediastinal	Total thyroidectomy; bilateral radical neck dissection; mediastinal dissection	No evidence of recurrence 2 yr. postoperatively

TABLE 1 (*Continued*)

SURGICAL REMOVAL OF THE SUPERIOR MEDIASTINAL LYMPHATICS FOR CANCER OF THE THYROID GLAND: SUMMARY OF CASES

Patient, Sex and Age (yr.)	Type of Growth	Location		Procedure	Follow-up
		Primary	Involved Nodes		
E. M., M, 26	Adenocarcinoma	Right lobe	Regional; mediastinal	Total thyroidectomy; right radical neck dissection; mediastinal dissection	No evidence of recurrence 2 yr. postoperatively
V. H., F, 14	Papillary adenocarcinoma	Left lobe	Bilateral cervical	Total thyroidectomy; bilateral radical neck dissection; mediastinal dissection	No evidence of recurrence 4 yr. postoperatively
M. M., F, 28	Papillary adenocarcinoma	Left lobe and isthmus	None	Total thyroidectomy; left deep cervical dissection; mediastinal dissection	No evidence of recurrence 1 yr. postoperatively
M. R., F, 52	Hürthle cell adenoma, malignant (?)	Right lobe	None	Total thyroidectomy; mediastinal dissection	No recurrence 7 mo. after operation

FIG. 2. Drawing made during course of surgical removal of metastases through dissection of the anterior-superior mediastinum. (From: McCORKLE, H. J. *West. J. Surg.*, 63: 324–333, 1955.)

FIG. 3. Illustration of structure exposed following removal of anterior-superior mediastinal contents. (From: McCORKLE, H. J. *West. J. Surg.*, 63: 324–333, 1955.)

superior mediastinum has been devised in the anatomy laboratories and applied to the surgical treatment of fifteen patients with cancer of the thyroid gland.

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DISCUSSION

Roy Cohn (San Francisco, Calif.): The extreme variation in the natural history of cancer of the thyroid no doubt accounts for the wide variations in opinion concerning the proper mode of surgical therapy. In criticizing the results of surgery for carcinoma of the thyroid, one should keep the pathologic picture in mind. In a study of the natural history of carcinoma of the thyroid, Frazell and Foote* found the following distribution in 301 cases:

301 CASES OF CANCER OF THE THYROID (1930–1946)

Type of Carcinoma	No. of Cases	Per cent
Papillary adenocarcinoma	139	46
Follicular and alveolar adenocarcinoma	22	7
Solid adenocarcinoma	56	19
Giant cell carcinoma	39	13
Hürthle cell carcinoma	27	9
Unclassified	18	6

* Frazell, E. L. and Foote, F. W., Jr. The natural history of thyroid cancer. *J. Clin. Endocrinol.*, 9: 1023, 1949.

Giant cell carcinomas are rapidly fatal, the average length of life being 4.8 months in this series, with no patient living for more than a year after the diagnosis was established. On the other hand, the papillary carcinomas appear at the other end of the scale; cases of recurrence thirty-five years later are reported. Even so, of the 139 patients in this series, 21 per cent were dead of the disease who were followed up five years or more.

In presenting their case for routine radical neck dissection in all cases of cancer of the thyroid, Morfit and Reimers* have offered the following data from Frazell and Foote:

PAPILLARY CANCER OF THE THYROID: NODE INVOLVEMENT BY LEVELS IN 182 SURGICAL SPECIMENS

Node Level	Clinically Positive Patients (115) (%)	Clinically Negative Patients (67) (%)
Submaxillary	9.7	1.5
Upper jugular	61	19
Mid-jugular	76	30
Low jugular	76	33
Spinal accessory	36	6
Juxtathyroid	32	13
Mediastinal	6	0

The question then appears to be the same as with other cancers: Is one justified in using the more extensive operative procedures? Since the operation advocated carries with it a low mortality and low morbidity rate, and since the careful studies of the natural history of even the most benign form of cancer of the thyroid show that it is a fatal disease in at least one-fifth of the cases, the answer would appear to be that we are so justified.

John F. Higginson (Portland, Ore.): I would like to congratulate Dr. McCorkle on his presentation. It has been quite interesting.

There are one or two points that perhaps might be helpful to others, which we found out the hard way.

A patient had an undiagnosed tumor presenting in the neck in the suprasternal notch involving the anterior mediastinum.

Exploration was performed with exactly the same type of thyroidectomy incision, with a drop "T," and sternal split, described by Dr. McCorkle.

I would like to suggest that, if necessary, this split can be carried with impunity completely

* Morfit, H. M. and Reimers, W. L. The emergence of standardization in the treatment of thyroid cancer. *Surgery*, 40: 1131, 1956.

through the sternum lengthwise, and not necessarily ended anywhere along the length of the sternum.

The closure is apparently, at least in our experience, no more difficult for the patient or the operating team.

The specimen removed was similar grossly to the ones that were presented by the author.

This tumor was in continuity with both lobes of the thyroid; therefore, both lobes were removed.

We could not tell what type of tumor it was from a biopsy specimen. Eventually it was identified as a parathyroid malignancy, after consensus of a number of pathologists throughout the country.

There are one or two points that perhaps might be mentioned further in addition to splitting the sternum all the way. It is not necessary to preserve all of the great vessels.

This tumor had to be removed in a manner as though one were raising a rug off the floor, starting from the lower end of the anterior mediastinum and taking pericardium and all of the areolar and fatty tissue that could be included in the specimen, including all the thymic tissue, the left innominate vein, a portion of the superior vena cava where the innominates meet, and the tissue on up to and including both of the thyroid lobes.

The patient experienced no discomfort from removal of one of the innominate veins; it is similar to excising a jugular vein in the neck.

One other point: it is not necessary ordinarily, I think, to worry about opening into one or both of the pleural cavities with the patient under standard intratracheal anesthesia. These cavities can be left open; in fact, with this type of procedure or with pericardiectomy for constrictive pericarditis, we simply insert the thoracostomy tubes from the mediastinum by way of stab wounds, and insert a hand into the chest on either side. This takes care of the situation adequately.

HORACE J. MCCORKLE (closing): Dr. Cohn has shown statistically that this procedure is not to be applied routinely to all patients with cancer of the thyroid.

The patients who have undergone mediastinal dissection for cancer of the thyroid were those in whom it was very likely, or almost certain, that the mediastinum was involved; this proved to be right in half the patients.

Dr. Higginson suggested that the sternum be split the entire length which, with this type of operation, gives a good result functionally and is just about as easy to repair as the type of incision I described.

Lymphocytic Thyroiditis in 757 Thyroid Operations

CARL A. PETERSON, M.D., *Palo Alto, California,* AND FREDERIC P. SHIDLER, M.D., *Menlo Park, California*

From the Department of Pathology, Palo Alto Hospital, Palo Alto California. This study was supported by the Medical Research Foundation for Palo Alto Hospital.

THIS study constitutes a résumé of certain pathologic findings in 757 thyroid operations performed at the Palo Alto Hospital during the years 1949 through 1956. The first part is concerned with the pathologic alterations of the surgical material, whether a biopsy, lobectomy or total thyroidectomy; the second part, with the clinical evaluation of the patients upon whom the operations were performed. For reasons discussed later, the study was undertaken independently by the two authors.

The purpose of the study was threefold: (1) to evaluate the relatively high incidence of lymphocytic thyroiditis in the Palo Alto area; (2) to study the pathologic and clinical relationship of struma lymphomatosa (Hashimoto's disease) with a group of glands showing lymphocytic infiltrate without significant follicular or epithelial changes; and (3) to ascertain the relationship of carcinoma of the thyroid gland to either or both forms of lymphocytic thyroiditis.

Terminology. The broad term, lymphocytic thyroiditis, as used in this paper, denotes a diffuse lymphocytic infiltrate of varying severity scattered throughout the parenchyma of the surgically resected tissue. Discrete lymphoid follicles with germinal centers were seen in all cases. Cases in which the infiltrate was of a localized type or had a reactive distribution, such as the lymphocytic infiltrate within or immediately adjacent to adenomas or carcinomas, were excluded. These reactive types of infiltrate were classified as grade 1 and, as noted in Figure 1, were not included in the data.

Only a small fraction of the cases fulfilled the usually accepted criteria for the diagnosis of struma lymphomatosa (Hashimoto's disease) in which, in addition to the lymphocytic infiltrate, atrophy of the thyroid follicles, compensatory hyperplasia and eosinophilic alteration of the epithelium is seen [1]. The primary finding in the majority of the cases was a varying degree of lymphocytic infiltrate, as noted in Figure 1. (No attempt was made to grade the follicular and epithelial alterations.) In some instances adenomas were found in association with the lymphocytic infiltrate, and in two instances carcinomas were present (specifically mentioned in section on thyroiditis associated with carcinoma).

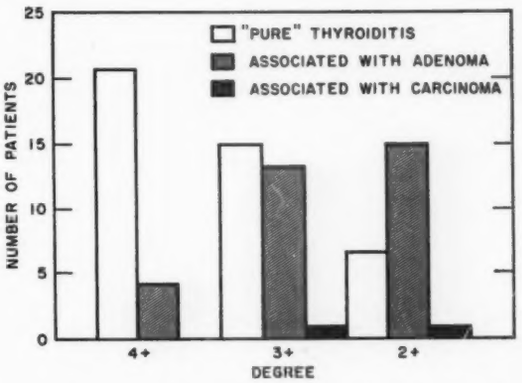

FIG. 1. Degree of severity of lymphocytic thyroiditis (seventy-seven cases).

Method of Study. The pathologic material from 757 thyroid operations was obtained from the files of the Pathology Department, restudied, and the data tabulated by one of us. (C. A. P.)

Six hundred seventy operations (88.5 per cent) were performed on women; eighty-seven operations (11.5 per cent) on men. The yearly number of operations performed during the last four years included in the study were more

than double those performed in the first four years, due largely to the marked increase of population in the area.

Incidence of Lymphocytic Thyroiditis. In the 757 operations, seventy-seven cases (10.1 per cent) of lymphocytic thyroiditis were

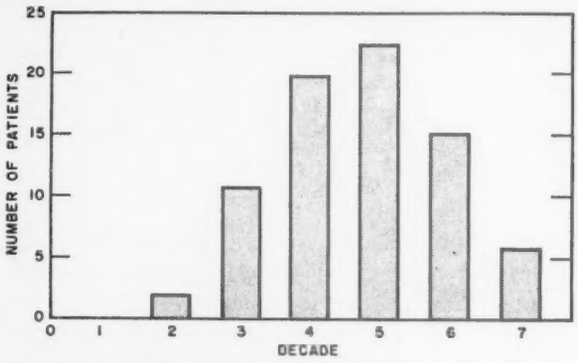

FIG. 2. Age distribution of patients with lymphocytic thyroiditis (seventy-seven cases).

included in the data after selection according to the previously mentioned criteria. Forty-three cases were free of other thyroid disorder (designated "pure" thyroiditis in Figure 1); thirty-two cases had associated benign adenomas of varying types. In the latter groups the entire specimen, beyond the adenomatous portion, showed varying degrees of lymphocytic infiltrate. Two cases of thyroid carcinoma, both of the papillary type, showed diffuse lymphocytic infiltrate in the non-malignant portions of the gland (to be discussed in detail in section on thyroiditis associated with carcinoma).

Sex Incidence. Seventy-five of these cases (98 per cent) occurred in females; two in males. One of the male patients had an associated carcinoma, the other a mild lymphocytic infiltrate, designated 2 plus. In neither instance were epithelial changes usually associated with Hashimoto's disease seen.

Age Distribution. Figure 2 shows the age distribution of the seventy-seven cases. The average age was 42.5 years, with the majority of cases occurring in the fifth decade. These findings are in close agreement with other reports [2].

Severity of the Thyroiditis. Figure 1 represents an attempt to evaluate the severity of the lymphocytic infiltrate within the various categories. The criteria used were as follows: Four plus—75 per cent or more of the parenchyma, replaced by lymphocytic infiltrate. (Figs. 3 and 4.) Three plus—approximately 50 to 75 per cent of the parenchyma replaced. (Fig. 5.) Two plus—approximately 25 per cent of the parenchyma infiltrated by lymphocytes. (Fig. 6.) The lymphocytic infiltrate, when not associated with other disorders, was, in general, more severe; when associated with adenomas, it was less intense.

Thyroiditis Associated with Carcinoma. Two instances of thyroiditis and carcinoma are included in the series; one in a twenty-five year old man, the other in a thirty-one year old woman. In both, the carcinoma was of papillary variety and confined to one lobe. The opposite lobe and parenchymal tissue not involved in the neoplastic process showed a diffuse lymphocytic infiltrate. However, the infiltrate, in each instance, had the distribution of a reactive type due to extensive calcification within the thyroid parenchyma. (Fig. 7.) No similar calcification was seen in any other case in the series. Crile and Fisher [3] reported two cases of papillary adenocarcinoma with lymphocytic infiltrate and parenchymal calcification which are almost identical to ours. Using the criteria previously mentioned, these cases should probably be excluded from this series, but since one of the objects of this study was to examine the relationship of thyroiditis and carcinoma, they were included.

Comparative Age Distribution. Figure 8 illustrates a comparison of the age distribution of the patients with carcinoma and those with lymphocytic thyroiditis studied in this series. There is one decade's difference in the average age as well as the period of maximum incidence.

Carcinoma. Thirty-three cases of carcinoma were found in the 757 operations, an over-all incidence of 4.3 per cent. Sex distribution revealed twenty-five cases in females and eight in males. Of the total operations on women (670), the incidence of carcinoma was 3.7 per cent, while of the eighty-seven operations on men, the incidence was 9.2 per cent.

The age distribution of the thirty-three patients is shown in Figure 9. The average age was 32.5 years.

Histologic Varieties of Carcinoma. The tumors were primarily of two types, papillary (sixteen cases) and follicular (fourteen cases). One was of the sclerosing type and two were of the undifferentiated variety. In twelve cases, lymph node metastases were present at the original operation. There was a significantly

FIG. 3. Struma lymphomatosa (Hashimoto's disease). Marked lymphocytic infiltrate (4+); follicular atrophy; epithelial hyperplasia with eosinophilia. No significant fibrosis.

FIG. 4. Struma lymphomatosa. Four plus lymphocytic infiltrate; marked localized epithelial hyperplasia with eosinophilia.

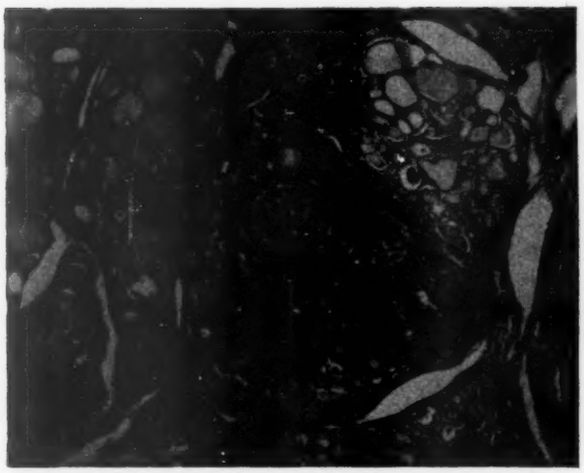

FIG. 5. Lymphocytic thyroiditis. Three plus lymphocytic infiltrate. No significant follicular atrophy or epithelial hyperplasia.

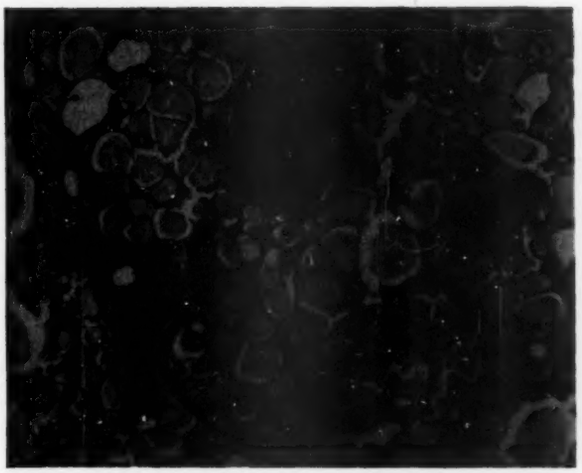

FIG. 6. Lymphocytic thyroiditis. Moderate lymphocytic infiltrate (2+). No follicular atrophy or epithelial hyperplasia.

higher percentage of malignant tumors in cases in which a clinical diagnosis of a single nodule had been made. Twenty-two per cent of such nodules were malignant in the male and 12 per cent in the female. (Note: In many instances the clinical diagnosis of a single nodule was not confirmed pathologically since the palpated mass was found to consist of two or more small nodules.)

CLINICAL ASPECTS OF LYMPHOCYTIC THYROIDITIS

The symptoms and clinical findings of these seventy-seven patients with lymphocytic thyroiditis could be ascertained in a general way from hospital records. The latter did not have the uniformity of observation of the patient or of the surgical pathology that would have been present if one surgeon had operated upon all of these patients. Instead, eight different surgeons had performed the operations.

All of these patients had goiters described as firm, sometimes diffuse, but with greater prominence of one lobe or of the isthmus. More than half of the patients had noted visible signs of neck swelling for months or years before operation. Usually the swelling had enlarged slightly or strikingly in the few weeks prior to operation. A very few patients had some feeling of pressure in the neck.

FIG. 7. Diffuse lymphocytic infiltrate and calcification in case of papillary carcinoma.

In slightly less than half of the seventy-seven patients there were no recorded symptoms; physicians had discovered the goiters in the course of physical examinations performed for other reasons. Those patients whose surgical specimen showed the severest forms of lymphocytic thyroiditis were under fifty years of age. None of the patients had preoperative myxedema. There were two males with this disease, one of whom had an associated carcinoma.

The few preoperative thyroid function tests recorded were the basal metabolic rate and protein-bound iodine. One patient with severe Hashimoto's disease had a slightly elevated basal metabolic rate and markedly elevated protein-bound iodine. All other values were low normal or normal. No protein flocculation, plasma protein or serum colloidal gold studies were recorded.

The correct tissue diagnosis was not made preoperatively in any patient. From physical examination or laboratory test, patients with struma lymphomatosa (as defined previously) could not be separated from patients with other forms of lymphocytic thyroiditis. All were operated upon in order to remove a nodular goiter or to prove or disprove the existence of carcinoma.

Nearly all patients were treated with bilateral subtotal thyroidectomy. Unilateral subtotal lobectomy was employed in some instances when a single adenoma was present. In a few operations only the isthmus was resected when both lobes seemed diffusely infiltrated.

The subsequent course of these patients after dismissal from the hospital is not known. Myxedema did not occur during the postoperative hospital stay. There was no surgical mortality.

COMMENTS

Incidence. The high incidence of diffuse lymphocytic infiltrate in thyroid tissue removed surgically may possibly indicate an unusual degree of this disease in the regional population. However, we believe the incidence rather reflects the difficulty of distinguishing nodular goiter from lymphocytic thyroiditis on the basis of clinical findings and preoperative laboratory tests. In this series the preoperative diagnosis nearly always was that of thyroid adenoma without hyperthyroidism.

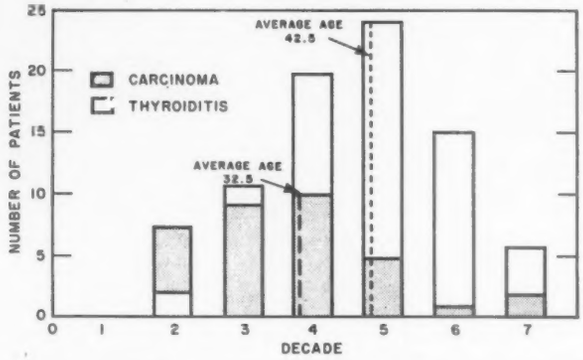

FIG. 8. Age distribution of patients with carcinoma of thyroid (thirty-three cases) and lymphocytic thyroiditis (seventy-seven cases); 757 operations.

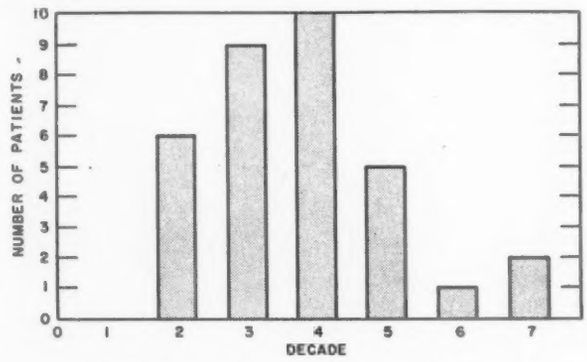

FIG. 9. Age distribution of patients with carcinoma (thirty-three cases).

At the operating table, surgeons rarely described uniform diffuse enlargement of the gland, but often found irregular enlargement of one or the other lobes which led them to believe they were dealing with adenomas or neoplasia as well as diffuse disease. This experience suggests that lymphocytic thyroiditis often appears indistinguishable clinically from

the nodular goiter of other thyroid disease. It is apparent that the diagnosis can be established only by direct examination of the thyroid gland. No needle biopsies of nodular goiters have been done at this hospital.

Relation of Lymphocytic Thyroiditis to Hashimoto's Disease. As noted previously, a large proportion of the cases included in this study did not show follicular atrophy and epithelial hyperplasia, usually regarded as criteria for the diagnosis of struma lymphomatosa or Hashimoto's disease; yet all of the cases, except for this microscopic difference, were strikingly similar. The age distribution of the cases in the two categories of lymphocytic thyroiditis was identical; the sex distribution was the same except for the one male included in the lymphocytic thyroiditis group. (Occasional cases of males showing Hashimoto's disease have also been described [4].)

In this study, as noted previously, the clinical survey was made by one of us (F. P. S.), without specific information regarding the nature of the pathologic alterations. The preoperative complaints, the operative findings and the postoperative course were identical in the two groups. At the conclusion of the study, with a knowledge of the two distinct variants of the lymphocytic thyroiditis, a concerted attempt was made to separate the two entities on a clinical basis. They could not be differentiated.

We, therefore, believe that the various types of diffuse lymphocytic infiltration of the thyroid gland, whether or not associated with follicular and epithelial changes, represent pathologic variants of a single disease process.

A parallel example may be cited in the case of cystic disease of the breast in which the mildest form consists of ectasia of ducts, but which may show pathologic variants from the formation of large cysts to a hyperplastic form in which the epithelial elements of the breast show marked proliferative changes such as fibrosing adenosis and intraductal papillomatosis.

A further interesting comparison with cystic disease of the breast is noted in Figure 10. The data concerning the cases of cystic mastitis was obtained from the surgical-pathologic material at the University of Minnesota [5], while the data concerning the lymphocytic thyroiditis was taken from this series. Although the number of cases compared in the diagram differs extensively, the similarity of the age incidence is striking. Since hormonal alterations associated with the premenopausal patient have been accepted to be the inciting factor in cystic mastitis, the often repeated theory of a hormonal etiology for lymphocytic thyroiditis would appear to have additional support. Just

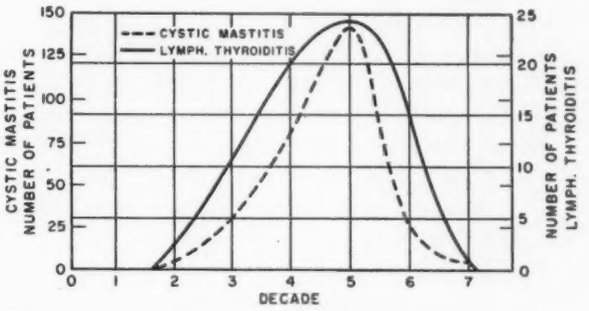

FIG. 10. Comparison of age incidence in cystic disease of breast and lymphocytic thyroiditis.

as an occasional case of cystic mastitis is reported prior to the third decade, so also an occasional case of lymphocytic thyroiditis appears. However, the maximum incidence in both diseases occurs during the period of most marked menopausal changes. Likewise, the sex distribution of lymphocytic thyroiditis, as generally agreed, shows a striking predominance for females, approximately 99 per cent.

CONCLUSIONS

1. Whatever the etiology, we, as well as other authors, believe that lymphocytic thyroiditis is not of inflammatory origin and can be separated histologically from acute, subacute, granulomatous and Riedel's types of thyroiditis.

2. The various microscopic variants of lymphocytic thyroiditis are different manifestations of a single disease process.

3. No increased incidence of carcinoma occurred in this series.

SUMMARY

1. Seven hundred fifty-seven thyroid operations were performed during the eight-year period, 1949 through 1956, at the Palo Alto Hospital, Palo Alto, California.

2. Seventy-seven (seventy-five female patients, two male) of the surgical specimens showed diffuse lymphocytic thyroiditis (10.1 per cent).

3. Lymphocytic thyroiditis occurred in rela-

tively "pure" form in forty-three patients, in association with adenomas in thirty-two patients, and in association with carcinoma in two patients. Cases in which the lymphocytic infiltrate was of a reactive type or of focal distribution were excluded from the data.

4. Only a small proportion of the cases showed follicular and epithelial alterations of the thyroid gland usually considered diagnostic of struma lymphomatosa (Hashimoto's disease).

5. Comparison between the cases of characteristic Hashimoto's variety with all others showed no significant differences in age group, sex incidence, clinical findings, preoperative diagnosis or postoperative course.

6. The relative age distribution of patients with cystic mastitis is similar to those with lymphocytic thyroiditis.

7. Thirty-three carcinomas were found in the 757 surgical specimens.

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DISCUSSION

Weldon K. Bullock (Los Angeles, Calif.): To make quite clear what we are trying to do, let us go back to the time of Hashimoto, when he described four cases before a surgical society similar to this one. To this day it is not quite clear to anyone what Hashimoto described. It is assumed that one of the cases was chronic thyroiditis.

Therefore, I think it is quite useless to try to propagate an eponymic disease such as Hashimoto's disease, when actually it is impossible to tell what Hashimoto described.

If we go a step further, those who would separate Hashimoto's disease from other chronic thyroiditis, because of the fact that in the latter plasma cells are found, are doing so on the assumption that the gland with Hashimoto's disease does not contain plasma cells. Yet, it has been clearly shown by Drs. Peterson and Shidler that clinically there is no difference. It is further stated by those who use the artificial separation that the oxyphilic changes in the follicular epithelium make the difference between Hashimoto's disease and other chronic thyroiditides. Here again, all one needs to do is read Hashimoto's original description to find that such minute details are totally lacking. Therefore, it can be seen from this study presented by Drs. Peterson and Shidler, as well as from the histologic point of view, that Hashimoto's struma or disease should be replaced by the term chronic thyroiditis.

At the Los Angeles County Hospital for the same period of time that this report was taken, 468 thyroidectomies were performed, in which there were twelve cases of thyroiditis. This is less than 1 per cent. Of these twelve cases, one was a tubercular thyroiditis, two were subacute thyroiditis, and one was acute suppurative strumitis. The latter was a suppurative adenomatous goiter, and I find that this is called strumitis rather than thyroiditis. The other eight cases were chronic thyroiditis of which, if you wanted to separate them on an artificial basis, only three were the so-called pure lymphoid thyroiditis, or what is termed Hashimoto's type.

This brought up the question of whether other hospitals in the area had a similar number of cases or were they in a higher percentage, as reported in the authors' paper.

At St. John's Hospital for the period from 1954 through 1956, cases of thyroiditis numbered approximately 8 per cent. It varied from 10 per cent to 7.5 per cent. Dr. George Hummer, Director of Laboratories, informs me that they grade these cases into grade I, II, III and IV, chronic thyroiditis, and do not separate them into Hashimoto's type.

At St. Luke's Hospital, Dr. Edward M. Butt, Director of Laboratories, likewise classifies this disease as chronic thyroiditis and does not use the term Hashimoto's disease. This laboratory, in 1947 to 1950, had eight cases in 201 thyroidectomies, which is only approximately 4 per cent; but in 1951 to 1953 there were eighty-nine thyroidectomies and fifteen cases of chronic thyroiditis, or almost 17 per cent. In 1954 and 1955 there were twenty-one cases in eighty-one thyroidectomies, or 25.9 per cent, and in 1956 they had ten cases in seventy-two thyroidectomies, or 7.2 per cent.

It would be very interesting to make a study of county institutions where the age group is much higher, and where, at the same time, one does not encounter the endocrine problems, such as cystic mastitis, that are seen in the private hospitals.

Of the 468 thyroidectomies performed at the Los Angeles County Hospital, the greatest number was in 1948. There has been a general decline in the number of people undergoing thyroidectomies. That is true at St. Luke's Hospital, as well as St.

Vincent's and St. John's, and I think it is somewhat general throughout the country.

I hope it has been made clear that all of these cases should be called chronic thyroiditis, rather than giving to some the eponymic name Hashimoto's disease.

WILLIAM P. KROGER (Los Angeles, Calif.): The problem of Hashimoto's disease seems confusing both to surgeons and pathologists alike. For many years I have listened to various discussions of the disease and evidently no one is clear as to the exact significance of the pathologic changes. Drs. Peterson and Shidler have presented a new and interesting concept, differing greatly from what has been offered in the past.

The gross appearance of Hashimoto's disease is unique and may be interesting to you. The tumor is typically large, bilateral, smooth and somewhat bosselated. The color is pale pink or yellowish, and the capsule is glistening.

We surveyed carefully the records of our thyroid patients from 1937 to 1957, with the exception of the years 1941 and 1942, and found ninety cases of Hashimoto's disease. An estimate of the entire twenty-year period would show about 100 cases, with an incidence of 3.9 per cent.

The cause of this rather mysterious disease remains obscure. In my experience, it has been a relatively innocuous condition and, aside from mild pressure symptoms, I have never known anyone to suffer ill effects. No carcinomas were observed in any of our cases. We are not in agreement with those who treat Hashimoto's disease with thyroid extract, or by radiation. We prefer the surgical approach; that is, radical subtotal thyroidectomy, leaving in perhaps 3 to 4 gm. of tissue on each side.

In studying the microscopic sections, I have been amazed at the almost complete absence of normal thyroid tissue. Oddly enough, few of our patients have become markedly hypothyroid, despite removal of the thyroid gland. They were all mildly hypothyroid—many before operation—but no myxedema developed in any patients.

DONALD F. BRAYTON (Los Angeles, Calif.): I am going to take a somewhat different approach to this situation.

Lymphocytic infiltration may occur in a variety of thyroid conditions, both benign and malignant.

The diagnosis of struma lymphomatosa, or Hashimoto's disease, however, rests mainly on the changes noted in the thyroid follicular cells, and in the colloid. These histologic findings in the goiter of a mildly hypothyroid or euthyroid female have led many observers to agree with Dr. Crile that Hashimoto's disease is not a chronic thyroiditis, but rather a primary thyroid failure with compensatory enlargement of the gland.

This enlargement is due mainly to hypertrophy and hyperplasia of the thyroid cells, rather than to the lymphocytic infiltration.

It has been estimated that this infiltration, spectacular though it is histologically, accounts for less than one-third of the weight of the goiter.

It is postulated that the hyperplastic follicular changes are due to a constant overstimulation of the gland by an increase in the pituitary thyrotropic hormone.

Crile and his group have shown fairly conclusively that this does occur by recording the radio-iodine uptake before and after a standard dose of thyroid-stimulating hormone.

In patients with struma lymphomatosa, or Hashimoto's disease, no increase in the radio-iodine uptake was apparent after a dose of thyroid-stimulating hormone. In contrast, patients with adenomatous non-toxic goiter, the condition most likely apparently to be confused with this lesion, showed an uptake after a dose of thyroid-stimulating hormone 50 to 100 per cent greater than that of the original tracer dose of I^{131}.

Evidently, in Hashimoto's disease the thyroid is already responding at peak level, probably due to constant intense pituitary stimulation.

The cause of the original thyroid failure responsible for the progression of events which leads to struma lymphomatosa is obscure.

Variations in estrogen levels, as with pregnancy and menopause, appear to play a role.

Of great interest is the recent work reported by Roitt and his associates. They became interested in the fact, previously known, that many patients with Hashimoto's disease have abnormally high serum gamma globulin levels which give rise to alterations in the thymol turbidity and colloidal gold flocculation tests.

They suspected that an immune reaction might be taking place, because of the inclusion of plasma cells within the infiltrate, and tested for precipitin reactions between the serum of patients with struma lymphomatosa and saline extracts of normal and thyrotoxic human thyroid glands.

The results showed positive precipitins in the serums of patients with Hashimoto's disease, and no precipitins in the serums of normal patients, or in those with other types of goiter.

This experiment tends to show that patients with struma lymphomatosa may have an auto-antibody against their own thyroglobulin.

Obviously much more work is necessary to confirm this bizarre hypothesis, but the test itself may well become useful in differentiating Hashimoto's struma with other types of goiter.

It seems logical and important to establish a clinical diagnosis of struma lymphomatosa, or Hashimoto's disease, since the removal of a thyroid so afflicted can do little but increase the patient's hypothyroidism.

There appears to be little evidence that there is an increased incidence of cancer in definitely established Hashimoto's disease, although it is well known that lymphoid invasion may occur in cancerous glands.

Crile's thyroid-stimulating hormone activation test and Roitt's precipitin test may eventually become within the reach of most clinical laboratories, and may then be of considerable aid in making the diagnosis clinically.

Needle biopsy is, of course, available now for those who have any faith in its accuracy.

In any event, a clinical trial of six to eight weeks of treatment with desiccated thyroid in adequate dosage appears to be logically indicated in those women suspected of having struma lymphomatosa.

It is well documented that this therapy will decrease the size of the goiter, often to cosmetically acceptable levels, and that the greatest amount of recession in size occurs within the first two months of treatment. If no such decrease in the size of the goiter is evident after an adequate clinical trial, then surgical excision should certainly be performed.

WILLIAM F. POLLOCK (Santa Monica, Calif.): I think it would be unwise for us to get involved with pathologists in a semantic discussion about lymphocytic thyroiditis, lymphadenoid goiter or Hashimoto's disease, but there is an abundance of evidence that whatever you call it it is associated with an increased incidence of carcinoma.

The studies over the last several years by Stuart Lindsay and his associates at the University of California on the association of benign and malignant neoplasms with what they call Hashimoto's thyroiditis are, I believe, quite significant.

In another report from the English literature Piercy has made the statement, the first time it has been said, I believe, that we should be performing thyroidectomies for Hashimoto's disease for the single indication that it is a premalignant lesion.

At Santa Monica Hospital and St. John's Hospital in the last five years, a total of fifty-two cases of Hashimoto's thyroiditis have been seen. In this series, six patients have had malignant thyroid neoplasms. Of these, five were carcinomas, of which four were papillary carcinomas. Of these four, three had metastases to the regional nodes.

The sixth case was a reticulum cell sarcoma, which I believe is the ninth or tenth case reported in the literature to date.

It is typical for a reticulum cell sarcoma of the thyroid to carry an extremely grave prognosis, and no patient survived beyond the second year.

Of the carcinomas that we saw, the average age of patients was thirty-six, and this is roughly in keeping with what Drs. Peterson and Shidler have shown.

The work mentioned by Dr. Brayton is of great interest.

Dr. John Payne, Professor of Surgery at the University of Buffalo, when he visited here last year, mentioned that he and Dr. Witebsky, in the Department of Bacteriology have, for the first time, produced a lesion which is identical with Hashimoto's thyroiditis in the experimental animal.

They have done this by preparing saline extracts of thyroid and injecting it into experimental animals. They believe that this is one of the best examples of true tissue specificity. The Hashimoto response can be obtained with thyroid tissue from many different animals.

I believe it is commonly stated that Hashimoto's thyroiditis does not occur in children, and it should be viewed with suspicion when it occurs in males.

Two years ago we presented before this society a report from the Los Angeles Children's Hospital on the nodular goiters we had seen in children. In that series were two patients who had Hashimoto's thyroiditis, one of them at the age of six.

CARL A. PETERSON (closing): When anyone opens up the subject of Hashimoto's disease, dissenting opinions, such as those expressed, can be expected.

Regarding Dr. Bullock's statements, I was pleased that he believed that the variations in microscopic appearance of lymphocytic thyroiditis represent pathologic variants of the same disease.

I also agree that pathologists must assume the major responsibility for the problems in terminology. From Dr. Hashimoto's early descriptions on down to Dr. Meisner's clear but restricted criteria at the present time, clinical and pathologic diagnoses have been at variance.

An example of such a problem among pathologists occurred recently when I wrote to a colleague in Minneapolis to obtain comparative data on the incidence of lymphocytic thyroiditis in the Midwest for the same period represented by our study. The reply stated, "What do you mean, pure Hashimoto's disease, or something else?" Thus we, as pathologists, are faced with this same problem of terminology, as are the surgeons.

The variation from year to year in the incidence of lymphocytic thyroiditis cases, mentioned by Dr. Bullock, was not seen in our series. This study was carried out in two periods of four years' duration each, and the incidence of lymphocytic thyroiditis in our area remained the same over the eight-year period within one-tenth of a percentage point.

Dr. Kroger stated that he had not seen any carcinomas in his large series of cases of Hashimoto's disease. I personally believe that the two cases of associated carcinoma and thyroiditis shown represent reactive phenomena. In our data, we attempted to exclude all lymphocytic infiltrations of reactive type, but for the purpose of completeness described these cases of carcinoma.

Dr. Brayton's remarks were well taken. I have read these papers and I think they are excellent.

We purposely have tried to avoid all discussions of etiology because of the marked difference of opinion, and because of the controversial nature of some of the recent work.

One interesting observation I would like to mention, however, is that in two cases of Addison's disease with complete destruction of the adrenal gland, one through tuberculosis and the other an idiopathic type, I have seen a very severe form of lymphocytic thyroiditis. Others have described the same finding. I do not know how this phenomenon would be explained by Dr. Crile s theory of the etiology of lymphocytic thyroiditis.

In regard to Dr. Pollock's remarks, the question of an increased incidence of carcinoma in lymphocytic thyroiditis or Hashimoto's disease is obviously very important.

I am well acquainted with Dr. Lindsay's findings.

Regarding our study, I can merely say that it covers an eight-year period, and had there been undiagnosed carcinomas during the early part of this series, I believe that we should have seen evidence of them by this time.

The Surgeon, the Urologist and the Patient with Abdominal Pain

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UNEXPLAINED abdominal pain presents one of the greatest challenges to diagnostic skill. When the cause or causes are of a surgical nature, surgeon and urologist alike may be confused and misled as to whether the fundamental disease is intraperitoneal or retroperitoneal. The general surgeon by training and inclination often tends to think of abdominal pain as being due to intraperitoneal disease and neglects the retroperitoneal structures. The urologist may think in terms of the urinary tract alone and neglect other retroperitoneal causes of pain or may confuse intraperitoneal disease with urinary tract disease. With increasing specialization in the fields of surgery, the true diagnosis may be overlooked by a specialist so intent on his own field that he neglects the consideration of fundamental facts which may enter into the problem. Bean has written, "One of the tragic by-products of an age of over-specialization has been the inability of the specialist to recognize his deficiencies outside his own field, and the corollary, his unwillingness to correct this deficiency by accepting advice from other specialists. An inevitable result of over-specialization is that as the focus narrows the intellectual requirements for success and achievement decline . . . " [2].

The purpose of this paper is to record a group of cases in which errors in diagnosis by the general surgeon or the urologic surgeon have led to incorrect handling of the patient with abdominal pain.

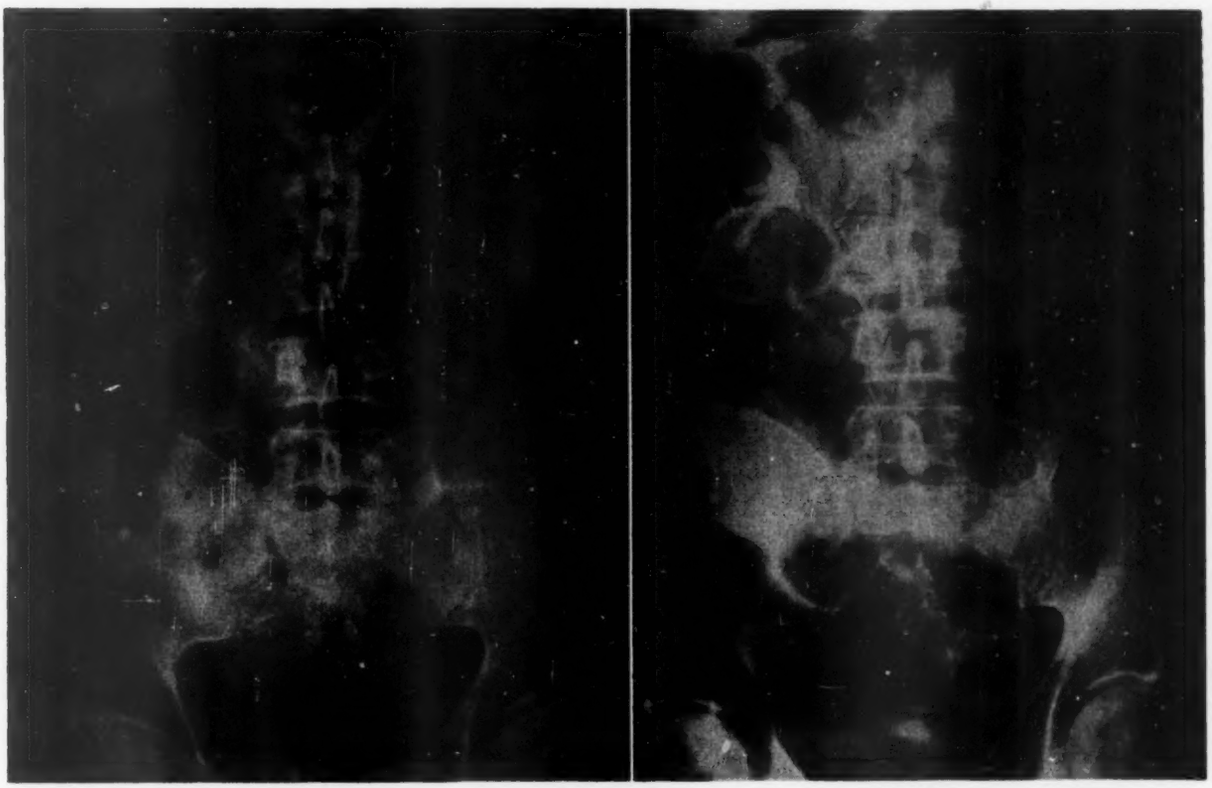
An appreciation of the regional anatomy of the abdominal organs and retroperitoneal structures and of the rich nerve supply of the kidneys and ureters with their abundant anastomoses to the celiac plexus helps in understanding why renal and ureteral pains may be indistinguishable from intraperitoneal, visceral pain [7,10]. In each case pain is usually caused by overdistention of a hollow viscus. Because of peristalsis and increased stretching usually due to obstruction, the pain is often cyclic and episodic. Obstructing lesions of the urinary tract may cause referred pain which mimics the pain of appendicitis, duodenal ulcer or gallstone. With acute ureteral obstruction there can be associated paralytic ileus which may be mistaken for mechanical bowel obstruction. On the other hand, intraperitoneal inflammatory conditions may produce colicky pain which simulates urinary tract obstructive pain and may produce red and white blood cells in the urine.

The problem of this confusion in diagnosis has interested us for many years, and a number of interesting and unusual cases have been collected demonstrating the difficulties that may arise. Some of the lesions encountered are listed here.

KIDNEY

Renal calculi have been mistaken for duodenal ulcer or appendicitis. Non-opaque renal calculi may cause ileus and lead to the diagnosis of an acute condition of the abdomen and exploration for presumed mechanical obstruction.

An alcoholic physician fell off of a ladder while painting. He was seized with severe, left-sided abdominal pain and was admitted to the hospital by way of the emergency room. The diagnosis was acute condition of the abdomen, possible ruptured viscus. A scout x-ray film on admission showed markedly dilated loops of bowel. The true cause of the difficulty was apparent on this film but was overlooked. (Fig. 1A.) Emergency exploration was



1A 1B

FIG. 1. Acute left pyonephrosis, secondary to obstructing calculus at left ureteropelvic junction. Note paralytic ileus. A, preoperative scout film before abdominal exploration for (?) acute condition of abdomen. B, postoperative intravenous urogram.

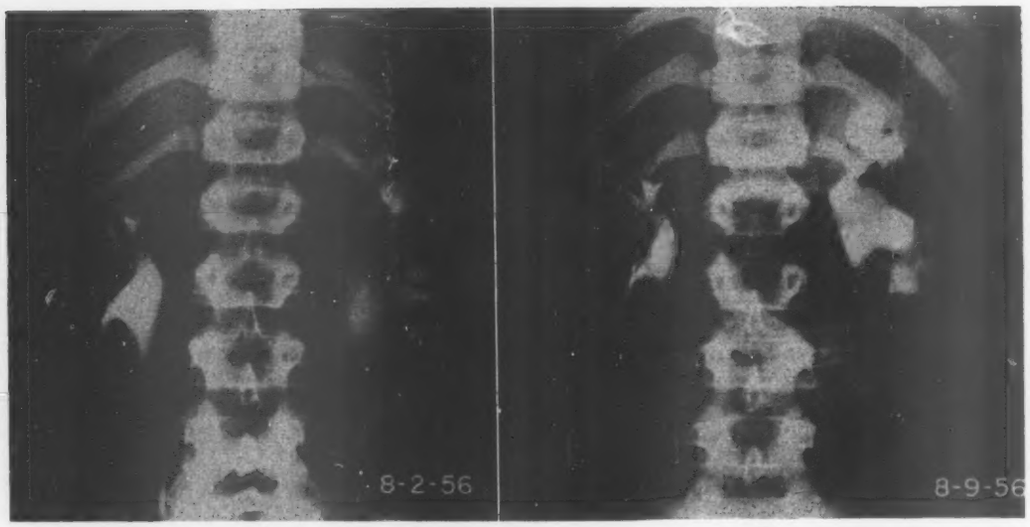
performed because of the possibility of a ruptured viscus. At exploration it was noted that the left kidney was large and swollen and that there was no intra-abdominal disease. The wound was closed and the patient was transferred to the urologic service. An intravenous urogram done the next day (Fig. 1B) confirmed the suspected postexploratory diagnosis. There was an obstructing ureteral calculus at the left ureteropelvic junction. The right upper urinary tract was normal. Because the patient was acutely ill, he nearly died before his condition warranted further surgery; nephrectomy of the left pyonephrosis was performed some days later.

If ureteral stone obstructing the left kidney had been suspected at the time of the exploratory operation, the surgeon might have removed the stone or at least have diverted the urine from the obstructed infected kidney, thereby giving the patient prompt relief of this severe difficulty. If an intravenous urogram had been made before exploratory surgery, it would have become apparent that the left kidney was non-functioning. This would have accomplished two things: it would have helped to make the correct diagnosis and would also have helped in the decision to perform nephrectomy which should have been done at the time of the exploration. The surgeon was right in not removing the left kidney without positive knowledge that there was a normally functioning right kidney.

Pyelonephritis may masquerade as appendicitis. We have seen a scrub nurse who underwent appendectomy because she had an attack of pain in the right lower quadrant of the abdomen due to pyelonephritis in the lower segment of a double kidney. It was only after the normal appendix had been removed that the correct diagnosis was later made.

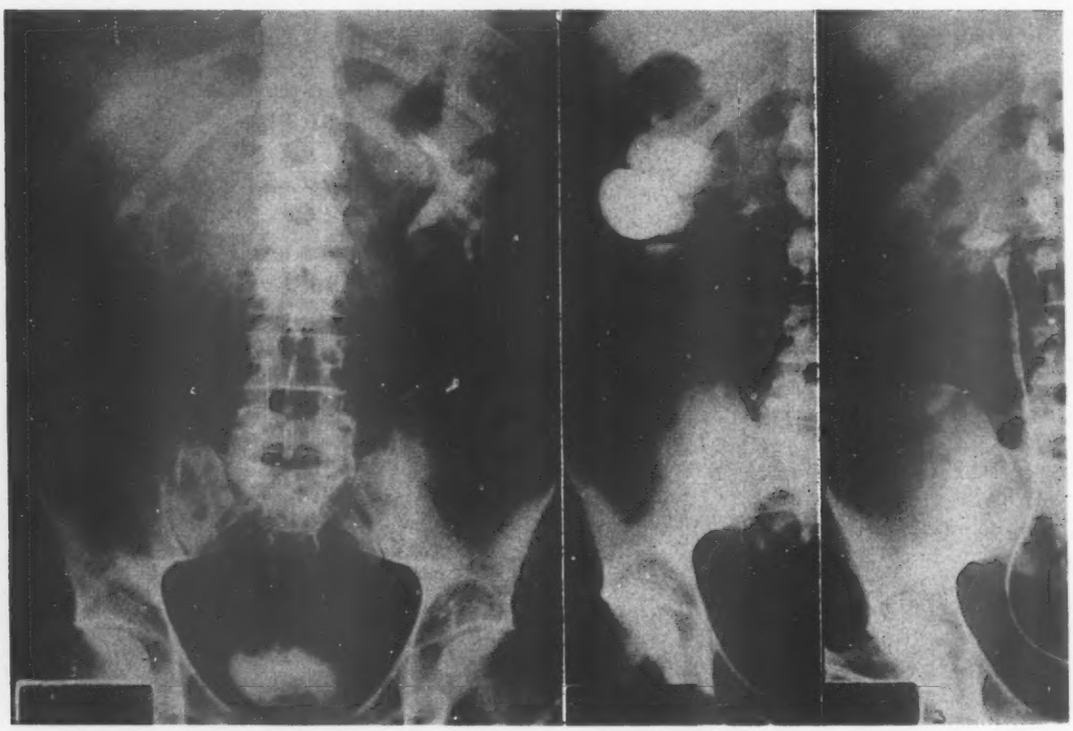
Renal infarction, although rare, has often been mistaken for appendicitis, particularly when the infarction occurs on the right side [1].

The pain of hydronephrosis with intermittent acute obstruction may suggest duodenal ulcer, or pyloric stenosis in young children. The fact that hydronephrosis may be intermittent should be appreciated. (Fig. 2.)



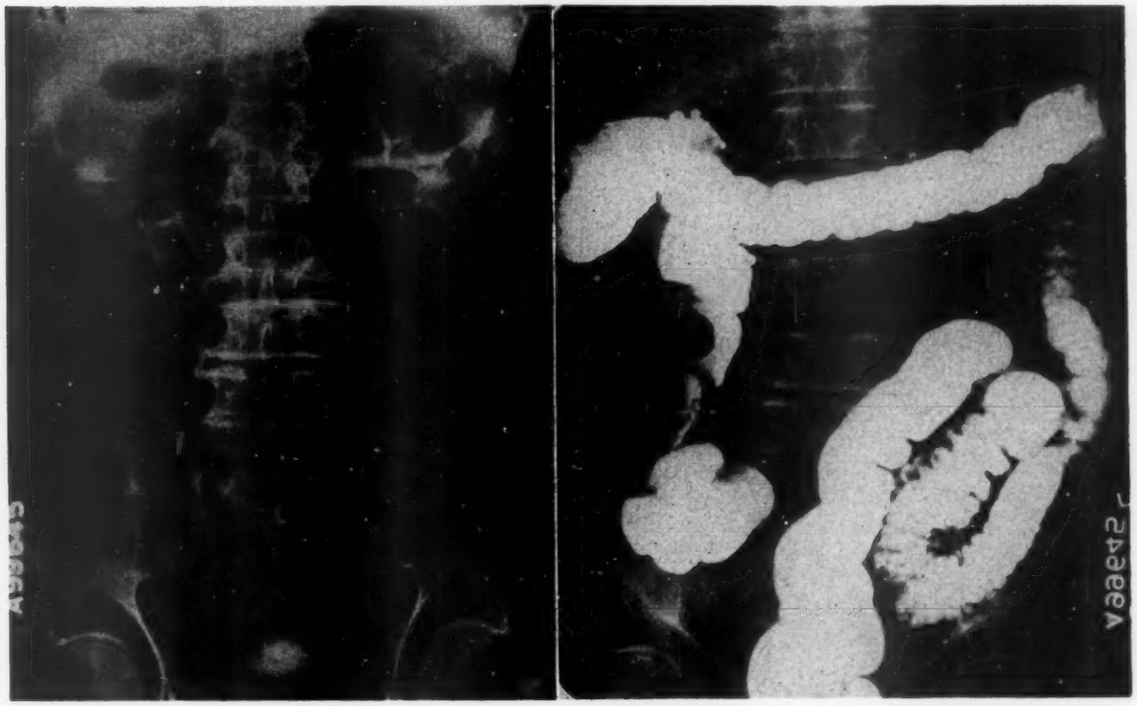
2A 2B

FIG. 2. Congenital intermittent left hydronephrosis. Lifelong history of intermittent episodes of acute abdominal pain, thought to be appendicitis. A, intravenous urogram made during acute attack. B, intravenous urogram one week later when patient was having no pain. The attacks of pain have disappeared since surgical correction of the obstruction.



3A 3B 3C

FIG. 3. Right pyonephrosis, secondary to congenital ureteropelvic junction obstruction. History of previous appendectomy and drainage of subphrenic abscess. Patient cured by removal of pyonephrosis. A, intravenous urogram showing non-function of right kidney. B, retrograde pyelogram. C, retrograde pyelogram.



4A 4B

FIG. 4. Hypernephroma invading cecum, mistakenly diagnosed as cecal neoplasm. Patient died within twenty-four hours after one-stage removal. A, intravenous urogram showing filling defect in right kidney. B, barium enema showing filling defect in cecum.

An infected hydronephrosis may be mistakenly diagnosed as subphrenic abscess, and acute hydronephrosis may be mistaken for mechanical ileus.

A thirty-six year old white man entered the accident room complaining of right-sided pain for one week. He stated that he "wished to have his subphrenic abscess drained again." The history revealed that about one year earlier he had suffered pain in the right flank and had an appendectomy. Two weeks later, when the pain continued, a diagnosis of right subphrenic abscess was made, and his right subphrenic area was drained. Following this, the wound discharged watery fluid for a long time and then finally closed. Thereafter, he continued to have intermittent pain in the right upper quadrant with chills and fever. Careful urologic x-ray study revealed a severe obstruction at the ureteropelvic junction on the right side with a large pyonephrosis above it. (Fig. 3.) When this was removed, the patient was cured of his difficulties.

Traumatic injuries to the gastrointestinal tract may also involve the kidneys. Renal trauma should be considered in any case of abdominal injury.

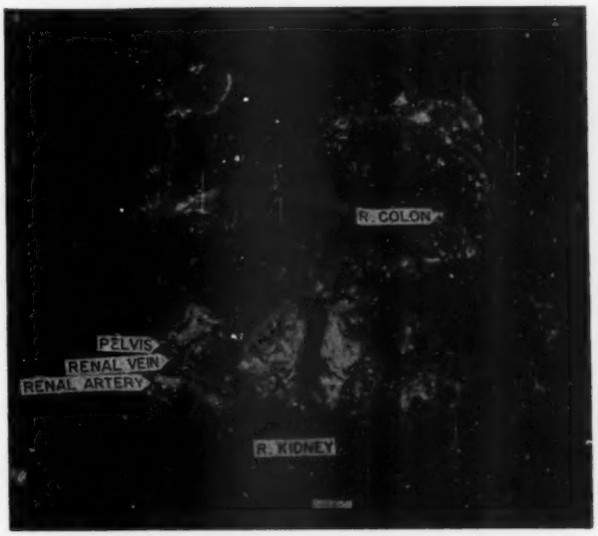
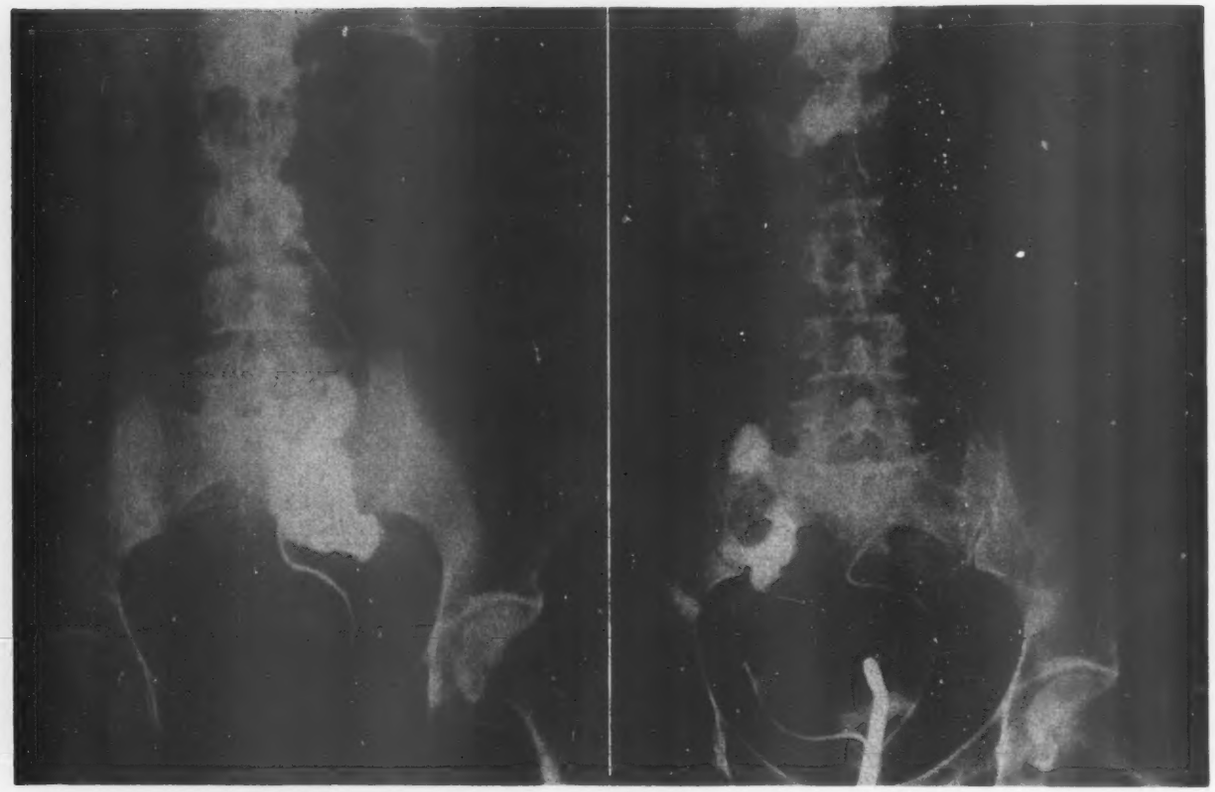


FIG. 5. Surgical specimen of hypernephroma invading cecum. (Compare with Figure 4.)

Right-sided hypernephroma has mistakenly been diagnosed as carcinoma of the ascending colon because of invasion of the cecum. (Figs. 4 and 5.)

Intermittent nephroptosis with pain from obstruction has been confused with intermit-



6A 6B

FIG. 6. Patient with bilateral pelvic kidneys and nephroileal fistula on right, secondary to calculus pyonephrosis. A, left pyelogram. B, right retrograde pyelogram showing filling of the fistula, terminal ileum and cecum. Patient cured by right nephrectomy and excision of segment of ileum.

ment bowel obstruction, and pain from a horseshoe kidney with ureteral obstruction has been confused with gastric ulcer.

Urologists have been confused by the following examples of *intraperitoneal* disease which were mistakenly thought to represent primary urinary tract difficulty on some occasions.

Gallstones have been mistaken for right renal calculi. Diverticulitis of the bowel may masquerade as ureteral calculus in transit. Appendiceal abscess and regional ileitis can produce ureteral obstruction and hydronephrosis with findings of microscopic hematuria and pyuria.

Pelvic inflammatory disease in the female may be confused with ureteral calculus. The pain from an ovarian cyst has led to gynecologic exploration and removal with the incidental finding of a silent hypernephroma discovered by palpation on the part of the alert gynecologic surgeon.

Carcinoma of the stomach with metastatic obstruction of the ureter has caused hydronephrosis and spontaneous renal rupture.

Cecal carcinoma has invaded the region of the right kidney and led to a mistaken urologic diagnosis of right hypernephroma.

A cyst of the tail of the pancreas or enlargement of the spleen has produced left renal displacement and pain thought at first to be primary renal disease.

ADRENAL

The adrenal glands are uncommonly the seat of pain, but there are occasional cases of intra-adrenal hemorrhage with production of a painful mass.

URETER

Ureteral calculus in transit may produce a paralytic ileus which has been confused with mechanical ureteral obstruction. (Fig. 1.)

Ureteral calculus is not uncommonly mistakenly diagnosed as appendicitis when it occurs on the right side. Hugh Cabot is quoted as saying that the most common operation for ureteral calculus is appendectomy.

Ureteral calculus and obstruction may cause

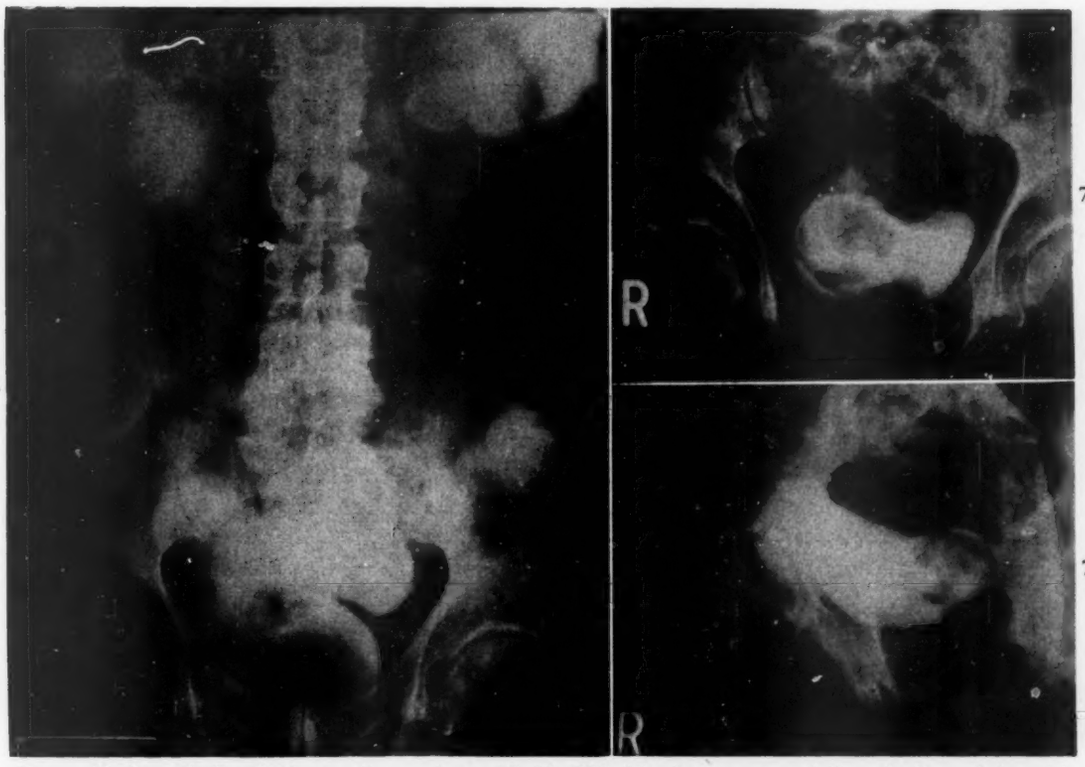


FIG. 7. Vesicocolic fistula due to abscess formed behind bladder from ruptured diverticulum of sigmoid. A, skiodan enema. B, cystogram. C, cystogram. Note filling defect in top of bladder. This characteristic sign made the diagnosis definite when cystoscopic examination confirmed it. Successful treatment was one-stage excision of involved segment of bowel and dome of bladder with primary re-anastomosis.

formation of a fistula between the urinary tract and the bowel. We have seen cases of nephrocolic and nephroileal fistulas due to this. (Fig. 6.)

Surgical trauma to the ureter following extensive gynecologic pelvic surgery or radical surgery on the left colon for malignancy may cause ureteral injury or obstruction which is slow to be recognized.

Cases of coexisting disease involving intraperitoneal structures and the urinary tract may be very confusing. In one such case the urologist was misled by a life-long history of multiple left-sided urinary calculi and the finding of urinary calculi in transit. The true cause of the patient's major difficulty, an advanced carcinoma of the cecum, was missed by the urologist at the initial examination. Another patient with known diverticulitis and cholelithiasis was admitted with an "acute abdomen" which finally proved to be due to an ureteral calculus in transit.

Ureteral tumors by their obstructive nature may produce gastrointestinal complaints leading to mistaken diagnosis of ulcer.

BLADDER

Bladder pain is usually recognized as such because it is low in the abdomen. One of the most common lower abdominal tumors is a distended bladder, and there are occasional cases in which exploration is performed for a lower abdominal mass which turns out to be a bladder distended from vesical neck obstruction.

In this series a patient with a huge diverticulum of the bladder had constipation and abdominal discomfort. A large filling defect was demonstrated by barium enema, but later proved to be due to a diverticulum of the bladder.

Stones in the bladder may cause intermittent pain but are usually recognized because of radiation of pain to the external genitalia and the invariable finding of blood in the urine.

Rupture of the bladder, if intraperitoneal, may cause an acute condition of the abdomen, which can lead to death if not properly handled.

Fistulas may develop between the bowel and the urinary tract. (Fig. 7.) They are usually due to abscess formation from regional ileitis

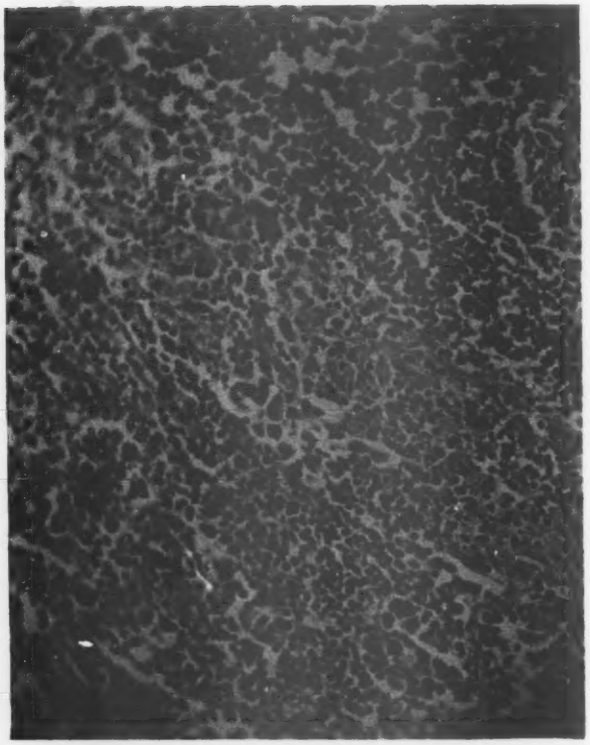


FIG. 8. Malignant tumor found in undescended testis because of abdominal pain in the region of the cryptorchid testis.

or diverticulitis of the colon. The abscess becomes walled off and then drains intermittently into the urinary tract, causing lower abdominal pain and intermittent pyuria and symptoms of urinary tract infection.

TESTIS

Pain in an undescended testis may be confusing. There are a few recorded cases of torsion or of epididymitis within an undescended testis. We have seen one patient in whom pain developed in the region of a cryptorchid testis. At operation this appeared to be normal, but was removed and found to contain advanced local malignancy. (Fig. 8.)

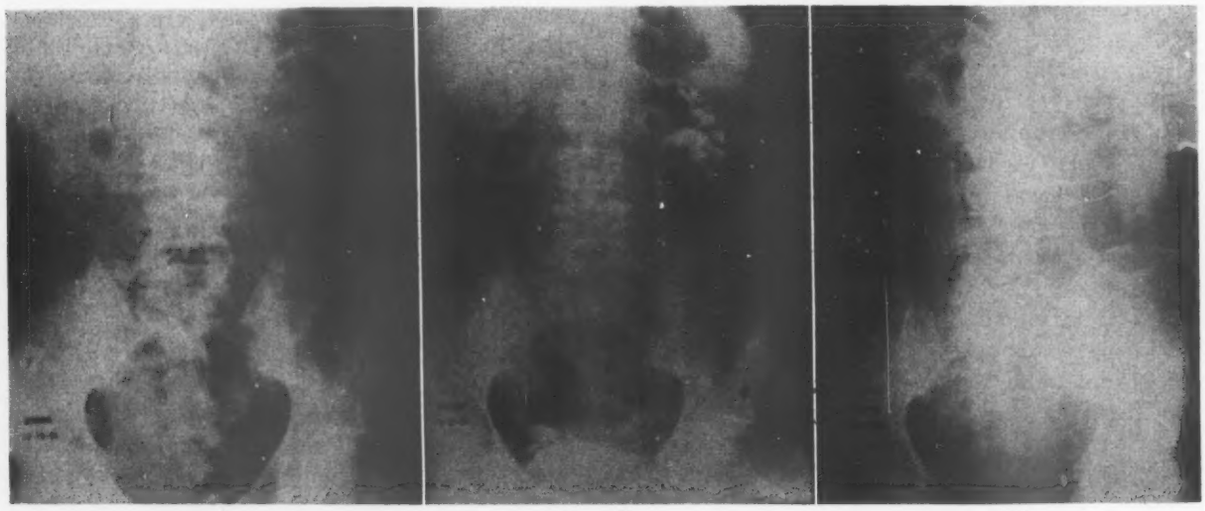
In this series there are several cases in which retroperitoneal lymph node metastases from a testicular neoplasm caused abdominal pain and led to surgical exploration before the correct diagnosis of primary tumor of the testis was appreciated.

RETROPERITONEAL CAUSES OF ABDOMINAL PAIN NOT RELATED TO THE URINARY TRACT

A dissecting aneurysm of the abdominal aorta may produce pain which is indistinguishable from that of a left ureteral calculus in transit. The added finding of mid-ureteral obstruction may still further cloud the picture. (Fig. 9.) Signs of shock and blood loss should suggest the true diagnosis.

A retroperitoneal hematoma may cause paralytic ileus and the findings of an acute condition of the abdomen.

Retroperitoneal abscess in the psoas, perinephric or subdiaphragmatic areas may cause ileus and be confused with intraperitoneal disease. Occasionally, an appendix ruptures



9A 9B 9C

FIG. 9. Dissecting aneurysm causing pain thought to be due to left ureteral calculus in transit. A, scout x-ray taken December 19th. B, intravenous pyelogram done December 21st. Note obstruction of mid-left ureter due to the aneurysm. Also, note beginning bulging of left psoas shadow. C, postoperative intravenous urogram after wiring of aneurysm. Patient died because diagnosis was not made early enough.

retroperitoneally and causes an abscess which is confused with perinephric abscess. We saw one such patient whose "perinephric abscess" was drained many times before a barium enema finally showed the presence of a fistula from the retroperitoneal ruptured appendix and led to surgical correction of the fundamental difficulty.

SUMMARY

All of these retroperitoneal causes of abdominal pain must be considered in the differential diagnosis of an acute condition of the abdomen. Important points in the study of the urinary tract before surgical exploration for unexplained abdominal pain are the following: (1) Accurate history and physical examination. (2) Careful microscopic examination of freshly voided urine in three separate glasses. (3) A "scout" roentgenogram of the abdomen to discover calculi or foreign bodies. (4) An intravenous urogram. (This is perhaps the most valuable of any single test in ruling the urinary tract in or out as a cause of unexplained abdominal pain. *It should always be employed when the diagnosis is in doubt.* Judging from the number of errors collected in this series, this simple and brief test should have been used in many more cases than is now the custom). (5) Cystoscopy, retrograde pyelography and, if necessary, retroperitoneal gas study and aortography may aid in the diagnosis of retroperitoneal disease.

These procedures should be freely employed as diagnostic measures whenever indicated. Retroperitoneal structures should always be included in consideration of the surgical causes of acute abdominal pain. The careful surgeon will insist on an intravenous urogram before abdominal exploration when the diagnosis is not absolutely certain. The knowledge gained concerning the condition of the urinary tract and the opposite kidney in cases of unilateral renal disease may prove lifesaving at the time of surgical exploration.

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DISCUSSION

Orville F. Grimes (San Francisco, Calif.): Here is a demonstration of case material which serves to remind us painfully of our own individual errors in diagnosis, judgement and treatment. The authors have discussed the many difficulties related to the efforts of general surgeons and urologists to solve the often manifold and confusing problems producing abdominal pain. Every surgeon has in his experience one or more near tragedies based on misinterpretation of symptoms and signs or on errors of omission. Such errors, although perhaps not justified, can nonetheless be explained by our desire to provide the necessary care of these patients without undue delay.

The essence of this paper is that in spite of the many inherent difficulties in the diagnosis of abdominal pain, the correct interpretation of signs and symptoms at their earliest stages into their different pathologic entities is most important. A very fine line can be drawn between the hazard to a patient's life by procrastination and the danger to life by erratic action. The sins of omission are considerable ones in the practice of abdominal surgery.

It is in this latter category that perhaps most of our mistakes are made. Perhaps Jenner's remarks can be paraphrased to say "more mistakes are made by not thinking than by not looking." In this respect, also, a return to fundamentals may be in order since the simple diagnostic tests of our forebears often are ignored or forgotten. Perhaps the pain on fist percussion over the lumbar region no longer has the significance that Dr. John B. Murphy placed on it in his discussion of physical signs with his students. Often, with nothing of confirmatory nature from the laboratory, with symptoms which are vague or non-localizing, the interpretation of such a simple physical sign

may indeed be the only one which aids greatly in a differential diagnosis. It is especially valuable in differentiating between the three common sites of lesions occurring acutely in the right side of the abdomen, namely, the gallbladder, the appendix and the kidney. When the test is positive on the left side of the abdomen, one can differentiate between pathologic conditions in the left half of the pancreas and in the kidney on that side.

The emphasis on diagnosis is always with us because only on a correct diagnosis will adequate and correct therapy be instituted. As Billroth told his students, "Ascertaining what is the matter with a patient is only the stepping stone to doing something of value for his improvement." In this intense desire to help the patient in abdominal distress, one must decide upon a course of diagnostic tests and investigative studies to pick the pathologic processes while they are still green, rather than to allow them to become ripe and drop beyond the realm of effective treatment. This is not to say, however, that waiting, and in a sense procrastination, is not without its value. The young student who enters the hospital with right-sided abdominal pain and symptoms of such severity to suggest possible appendicitis, but who in the judgement of the surgeon can be observed for a few hours or a day or so, may be helped immeasurably by such a delay, especially if the mild jaundice of hepatitis becomes evident after forty-eight hours of hospitalization. It is unfortunate, perhaps, that the architect placed so many important structures in and about one body cavity. Since it is reasonable to assume that little, if any, alterations can be expected, those of us who administer to those with the often confusing array of abdominal symptoms must accept that our greatest efforts will be toward pinpointing an exact diagnosis. Once such is made, the surgical treatment of an exact condition is indeed quite simple.

WILLIAM H. SNYDER (Los Angeles, Calif.): I have enjoyed this paper very much.

There is one urologic condition that the authors did not have a chance to mention. During the first year of the war, while working in a Station Hospital, we saw a fairly large number of these patients. I should like to mention this condition because I have seen it occasionally since then and think it is worth while bringing it to your attention. The condition to which I refer is acute seminal vesiculitis.

In this age group of young adults, during one year's observation, we saw nineteen such cases. All were admitted with a diagnosis of acute appendicitis. Although the symptoms are quite alike in these two conditions, the differential diagnosis can be made on rectal examination by feeling an acutely tender seminal vesicle. Therefore I bring this to your attention not because it will be frequent but because occasionally it will be very worth while to remember it.

LEON GOLDMAN (San Francisco, Calif.): There is another aspect of this subject that I think is worthy of mention, and it concerns surgical training.

It was mentioned that this problem arises because a man in one special field of surgery is not cognizant of the problems that arise in a neighboring field. At the present time, young men going through their training in surgery are greatly impressed by the advances in cardiac surgery, e.g., some of the aortectomies that have been discussed, and the more dramatic technics. They tend to shun spending time in other services, particularly urology.

I am sure the mistakes that the authors brought before us and reminded us of often can be prevented during the training period. It is during the period of training in a field such as urology that the basic precepts are laid down in the young and embryonic general surgeon. At that time one is impressed with the extensiveness of pathologic lesions of the retroperitoneal area and the absence or paucity of symptoms which, when they are brought to light, may be misinterpreted by the general surgeon.

Even though the training in general surgery seems, as the years go by, to allot more and more time to other allied fields than it may to general surgery itself, there is a need to preserve this rounded period of training.

WILLIAM F. POLLOCK (Santa Monica, Calif.): The authors made the point that two of the errors committed were those of failure to divert the urinary stream. One patient died, the other did not. In both cases it was believed that the general surgeon who opened the abdomen should have diverted the urinary stream.

I wonder if most general surgeons are aware of the indications for nephrostomy? I wonder also about the common problem of the kidney that is injured at the same time a spleen is ruptured?

I have had the experience on three occasions of opening an abdomen for a ruptured spleen and finding that the kidney was ruptured also.

Urologists to whom I have presented these cases (and in one instance a urologist was with me on the case) said that they do not routinely remove the kidney if it is shattered by the blow that ruptured the spleen. They have a very conservative attitude about removing damaged kidneys. They believe that they can be removed at a time and place of election, and that many patients with fractured kidneys will survive.

I wonder if Dr. Goodwin would comment on the indications for nephrectomy in the damaged kidney at the time the abdomen is opened for, let us say, a ruptured spleen or a perforated hollow viscus.

WILLARD E. GOODWIN (closing): I could not agree more with what Dr. Goldman had to say about the necessity for including training in the specialties as a part of general surgical training. I

certainly am delighted that he expressed himself thusly on this important point.

To try to answer your question, Dr. Pollock, we have a very conservative attitude concerning nephrectomy, unless there are positive indications and the other kidney is healthy and capable of supporting life. I believe that every patient with abdominal trauma and many others with possible urologic problems should have an intravenous urogram before surgical exploration. If the surgeon is faced with the decision of performing nephrectomy at the operating table, he may be in deep trouble if he does not know whether the other kidney is all right. Under these circumstances an intravenous urogram is a must.

The positive indications for removing a kidney are uncontrollable hemorrhage or irreparable damage to its blood supply. Severe calculus disease, pyonephrosis, advanced hydronephrosis and neoplasm are other indications, but one must be sure of the condition of the remaining side first. When in doubt, in cases of obstructive disease, it may be a lifesaving measure to divert the urine to the skin by way of nephrostomy or pyelostomy tube drainage placed extraperitoneally.

I want to thank the other discussants. I wish particularly to thank Dr. Snyder for mentioning the testis and the seminal vesicles; I wanted to discuss this but did not have time.

In this series there were three cases in which the testis was a cause of abdominal pain. One patient had an undescended testis and hernia with pain. When the cryptorchid testis was removed, it was found to contain a seminoma.

The other two patients were admitted to the surgical service. One underwent herniorrhaphy; later, the testis became swollen and tender and there was a wound infection. After this patient was observed for five months, an inoperable cancer of the affected testis had developed.

The third case was particularly striking because the patient was operated upon twice before the diagnosis of a tumor of the testis was made from biopsy of a retroperitoneal node; all this in spite of the fact that the tumor of the testis was easily palpable on physical examination. Exploration was performed at one hospital for an abdominal mass and pain. A huge pulsating mass was found in the retroperitoneum and the surgeon thought it was an aneurysm. The patient was transferred to the Veterans Hospital with the diagnosis of aortic aneurysm. The vascular surgeons quickly got out their grafts and went after the mass again. It was not an aneurysm. A biopsy specimen at the second operation showed a metastatic tumor of the testis. Then the testis was finally examined and found to contain an obvious tumor.

One final retroperitoneal cause of abdominal pain that should be mentioned is dissecting aneurysm. The urologist as well as the general surgeon should be aware of this distressing and unrelenting type of pain which may masquerade as an ureteral calculus. Dr. Turner and I have seen two patients who died of dissecting aneurysm under these circumstances. In each case a surgeon had made the diagnosis of ureteral calculus and sent the patient to a urologic service. There the urologists agreed with the diagnosis because it seemed so typical of ureteral pain. The distribution of pain was, of course, left-sided and episodic, and an obstructive change in the pyelograms and microscopic hematuria further suggested a ureteral calculus. The patients died before our eyes when the aneurysms finally gave way. This condition must not be overlooked. A telltale sign is a change in the left psoas shadow in repeated abdominal roentgenograms.

Surgical Treatment of the "Dumping Syndrome"

AN EVALUATION BY CHANGES IN PLASMA VOLUME, PERIPHERAL BLOOD FLOW AND ELECTROENCEPHALOGRAPHIC PATTERNS

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PARTIAL gastrectomy is the most widely used and well established method for surgical treatment of chronic peptic ulceration. However, certain undesirable features sometimes follow partial gastrectomy which tend to limit the scope of the operation. Chief among these is the so-called "dumping syndrome" which is a symptom complex that appears promptly after eating and is characterized by nausea, weakness, palpitation, sweating, varying degrees of syncope, a sensation of warmth and sometimes diarrhea. There has been much speculation about the cause of the dumping syndrome and some have believed that it was largely a psychogenic or functional type of disturbance. However, Roberts et al. [*13*] have shown that a fall in plasma volume occurs during the height of dumping attacks. In addition, Smith [*14*] and Roberts et al. [*12*] demonstrated that electrocardiographic changes are present during dumping attacks; these changes simulate the electrocardiographic findings seen in coronary artery insufficiency. It was recently shown by Hinshaw et al. [*5*] that definite increases in peripheral blood flow occur in patients with severe dumping attacks. They also demonstrated electroencephalographic changes during dumping episodes. They postulated that severe "dumpers" are persons who have abnormal vascular homeostatic responses, and consequently severe systemic or vasomotor symptoms develop when sudden reductions in blood volume occur. They also believe that the systemic symptoms of dumping such as weakness, syncope, warmth and sweating with their accompanying electrocardiographic and electroencephalographic changes may be associated with decreased cerebral and coronary blood flow. Studies are in progress to clarify this problem further.

It is generally agreed that severe clinical dumping is closely related to very rapid or precipitate gastric emptying and it may follow any operative procedure on the stomach in which the pyloric sphincter mechanism has been destroyed. Dumping symptoms may follow any type of gastric resection; however, they are most frequent following total gastrectomy, but may even appear after gastroenterostomy alone. Such symptoms are known to occur after any of the various modifications of partial gastrectomy. However, there is reason to believe that the incidence of severe dumping is less following the Billroth I (gastroduodenostomy) modification than it is following the Billroth II (gastrojejunostomy) reconstruction. Kanar [*6*], reporting on 451 patients who had Billroth I type resections for benign disease, stated that eighteen patients had mild dumping symptoms, but only three patients had incapacitating symptoms, an incidence of 1.4 per cent for the latter group. Gastric emptying is thought to be somewhat slower after the Billroth I operation than after the Billroth II. It is believed by many that the size of the stoma has no effect on the emptying time of the stomach. Kennedy, Reynolds and Cantor [*7*] have suggested that the factor which controls the emptying time is not stomal size, but the size of the jejunal lumen. This, however, would

not necessarily apply if the size of the stoma were smaller than the jejunal lumen, and it does not take into consideration any possible sphincteric action of the stoma itself.

Porter and Claman [*11*], have advocated the use of a small stoma in the Billroth II gastric resection. They believe it promotes sphincteric action and tends to delay the emptying time of the stomach, consequently reducing the incidence of the dumping syndrome. They advocate the use of a stoma 2.5 cm. in size, which is considerably smaller than that used in the customary Hofmeister-Finsterer Billroth II gastric resection. They operated upon 118 patients using the small stoma technic and reported an incidence of dumping comparable to that reported for Billroth I operations by Kanar et al. [*6*]. Porter and Claman [*11*] present radiographic follow-up data on patients to show that the emptying time is delayed by their small stoma method. Dragstedt [*3*] has also noted a decrease in the incidence of dumping symptoms when small-sized gastrojejunostomies are used in conjunction with vagotomy.

The problem of surgical treatment of the dumping syndrome has been further complicated by the fact that it has been difficult to differentiate between patients whose symptoms had an actual physiologic basis and patients whose symptoms were chiefly neurotic or functional in nature. During recent years the physiologic basis of the dumping syndrome has become better defined so that it is now possible to identify the patients with true dumping symptoms by giving them test meals which reproduce the symptoms and cause definite measurable physiologic changes. The real dumper is identified not only by clinical symptoms, weight loss and rapid gastric emptying but also by changes in plasma volume, peripheral blood flow and electrocardiographic and electroencephalographic tracings.

Attempts at surgical correction have largely centered around two approaches. Some have narrowed gastrojejunostomy stomas hoping to delay gastric emptying, while others have converted gastrojejunostomies to gastroduodenostomies. The conversion of Billroth II operations to Billroth I for relief of postgastrectomy symptoms has been particularly advocated by Perman [*10*] in Stockholm. Mathewson [*9*] recently reported two patients with postgastrectomy symptoms who were benefited by conversion from a gastrojejunostomy to a gastroduodenostomy. Woodward, Desser and Gasster [*16*] successfully treated three patients with severe dumping by converting gastrojejunal to gastroduodenal anastomoses and by narrowing the previously large stomas.

MATERIALS AND METHODS

This study is based on five patients with severe dumping symptoms who were treated by conversion of Billroth II gastric resections to a Billroth I end-to-side Finney-von Haberer type modified by the use of a small stoma. Certain observations were made before and after surgical conversion. These consisted of plasma volume determinations in four patients (using Gibson's indirect modification of the Evans blue dye method) [*4,13*]; peripheral blood flow studies and skin temperature determinations in one patient (using the Winsor pneumoplethysmograph [*5,15*]; electroencephalographic tracings in one patient; and comparative x-rays showing differences in gastric emptying time in another. The physiologic observations were made during dumping attacks induced with oral test meals of 150 cc. of 50 per cent glucose.

Exactly the same conversion operation was performed in each of the five cases. The previously made large stoma gastrojejunostomies were dismantled and jejunal continuity reestablished. The open end of the stomach was then closed from the lesser curvature toward the greater curvature side with a 2.5 cm. area on the greater curvature being reserved for subsequent anastomosis. The peritoneal reflection lateral to the duodenum was incised to facilitate extensive mobilization (Kocher) so that it was possible to approximate the gastric remnant and the duodenum without difficulty. The previously closed duodenal stump, which in most instances had been sutured into the pancreatic capsule, was left undisturbed. The anastomosis was made between the stomach remnant and the side of the duodenum with a lumen just large enough to admit the tip of the index finger. Figure 1 illustrates this small stoma, end-to-side gastroduodenostomy. The postoperative care of the patients did not differ in any way from that of the usual gastric resection.

CASE REPORTS

CASE 1. B. L. S., a forty-six year old woman had severe incapacitating symptoms consisting of

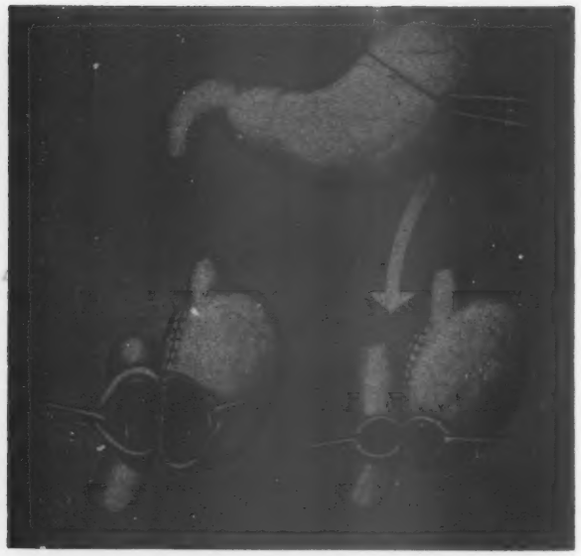

FIG. 1. A, the 2.5 cm. area on the greater curvature which is used in making a small stoma anastomosis. B, the partially completed small stoma anastomosis performed as an end-to-side gastroduodenostomy (Finney-von Haberer). C, an end-to-side anastomosis of conventional size for comparison with the small stoma anastomosis shown in (B).

nausea, a feeling of warmth, weakness and syncope beginning fifteen minutes after each meal. She was forced to lie down after meals to obtain partial relief. Her dumping problem began after a large stoma, Billroth II gastric resection was performed three years previously for duodenal ulcer disease.

TABLE I

SUMMARY OF PHYSIOLOGIC VALUES BEFORE AND AFTER SURGICAL REVISION IN CASE I

Data	Values in Induced "Dumping" Attack		Values after Surgical Revision	
	Before	During	Before	During
Plasma volume (cc.)	2,450	1,740 (710 shift)	2,500	2,250 (250 shift)
Peripheral blood flow (finger) (cu. ml./sec.)	3.5	32	4	6.5
Peripheral skin temperature (°c.)	31	35	31	32
Clinical symptoms	None	Severe	None	Slight nausea only

She was given a test meal which produced a severe clinical dumping attack. (Table I.) This attack was accompanied by a maximum fall of 710 cc. in plasma volume; a tenfold increase in peripheral blood flow with a 4°c. rise in skin temperature; and a definite abnormality in the electroencephalogram. (Fig. 2.) Surgical conversion to a small stoma, end-to-side Billroth I was effected, following which her symptoms disappeared; she now eats an essentially normal diet and has consistently gained weight. Two months later the objective physiologic studies were repeated after giving the patient an identical test meal. At this time the plasma volume fall was only 250 cc. as compared with 710 cc. before. The peripheral blood flow increased only one and a half times as compared with a tenfold increase before, and the skin temperature rose only 1°c. as contrasted with the previous 4 degree rise. (Table I.) Whereas the test meal had previously induced a severe clinical reaction, no symptoms were produced during this second study. In addition, the electroencephalogram remained normal at this time. (Fig. 3.)

TABLE II

SUMMARY OF CHANGES IN PLASMA VOLUME AND CLINICAL SYMPTOMS BEFORE AND AFTER SURGICAL REVISION IN CASES II, III AND IV

Case No.	Plasma Volume, Maximum Drop (cc.)	Clinical Symptoms	Plasma Volume, Maximum Drop (cc.)	Clinical Symptoms
	Before	Before	After	After
II	735	Severe weight loss; fullness	275	None
III	700	Severe dumping	270	None
IV	675	Severe dumping	250	None

CASE II. J. L., a sixty-nine year old man, had immediate postprandial distress consisting chiefly of epigastric fullness. This followed a Billroth II, large stoma, Polya type gastric resection performed five years previously for duodenal ulcer disease. These symptoms caused the patient to restrict his food intake closely and resulted in a severe weight loss problem. He did not complain of syncope, sweating or weakness. A test meal reproduced the epigastric fullness and nausea, and a maximum fall of 735 cc. in plasma volume occurred. (Table II.) Following surgical conversion to a small stoma, end-to-side Billroth I the studies were repeated under identical circumstances. No symptoms were produced and the fall in plasma volume was only 275 cc. at this second study as compared with 735 cc. before. Since his reoperation seven months ago, this patient has gained 20 pounds in weight, eats normal meals without distress and considers himself to be well.

CASE III. E. H., a twenty-nine year old woman, had typical, incapacitating dumping attacks consisting of nausea, warmth, syncope, sweating and weakness coming on immediately after eating,

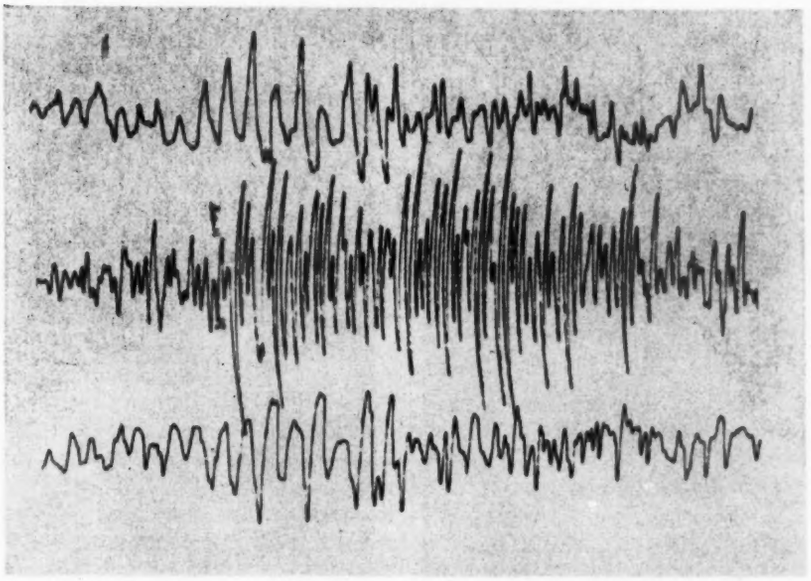

FIG. 2. Case 1. Electroencephalographic tracing showing cerebral dysrhythmia during a dumping attack.

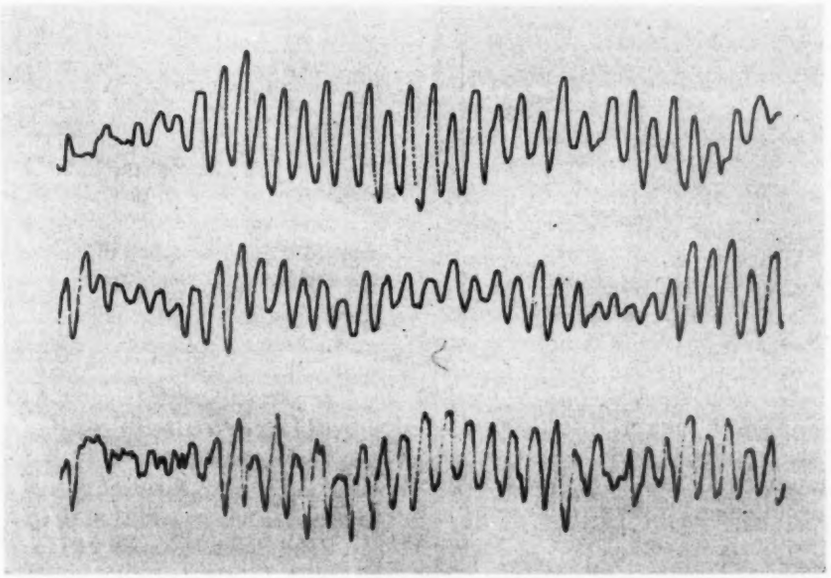

FIG. 3. Case 1. A normal-appearing electroencephalographic tracing made during a controlled test meal following surgical revision.

partially relieved by lying down. These symptoms with consistent weight loss followed a large stoma, Billroth II gastric resection performed eighteen months before for duodenal ulcer disease. A test meal was given which reproduced her dumping symptoms and a plasma volume fall of 700 cc. (Table II.) Following surgical conversion to a small stoma, end-to-side Billroth I, her dumping symptoms disappeared and have not returned during an eight-month follow-up period. A repeat study with a similar test meal four months after reoperation showed only minimal clinical symptoms and a maximum plasma volume fall of 270 cc. as compared with 700 cc. before the corrective operation.

CASE IV. P. S., a thirty-one year old woman, had severe dumping symptoms consisting of nausea, warmth, sweating and weakness beginning ten minutes after each meal. These attacks began following a large stoma, Polya type Billroth II operation performed fifteen months earlier for benign gastric ulcer. She progressively lost weight following surgery and found it necessary to lie down after each meal for partial relief of her symp-

FIG. 4. Case v. Roentgenogram showing excessively rapid gastric emptying and jejunal dilatation before surgical revision.

FIG. 5. Case v. Roentgenogram taken four months after surgical revision demonstrates delayed gastric emptying into the duodenum. The time of exposure following barium ingestion is comparable in Figures 4 and 5.

toms. She was given a test meal which produced a typical clinical attack of dumping accompanied by a fall of 675 cc. in plasma volume. (Table II.) Surgical conversion to a small stoma, end-to-side Billroth I was effected, following which her symptoms completely disappeared. She now eats normally and has consistently gained weight. A similar test meal was given seven months after her reoperation; at this time it failed to produce any symptoms of dumping and the maximum plasma volume fall was 250 cc. as compared with 675 cc. previously.

CASE V. This forty-five year old woman had severe, persistent postprandial distress for twelve years following a Billroth II, Polya type gastric resection. Her symptoms consisted of nausea, warmth, sweating and weakness of mild to moderate severity coming on fifteen minutes after eating, but followed by severe watery diarrhea. The diarrhea was her most intolerable symptom. A test meal produced the symptoms including the diarrhea. No blood volume studies were obtained, but roentgenographic studies showed extremely rapid gastric emptying. (Fig. 4.) Surgical conversion to a small stoma, end-to-side Billroth I was effected, following which her symptoms including the diarrhea have greatly improved. Repeat roentgenographic studies of the stomach four months later showed a definite delay in gastric emptying time as compared with the studies made before her reoperation. (Fig. 5.)

RESULTS AND COMMENTS

The clinical results obtained by the conversion of large stoma, Billroth II gastric resections to a small stoma, end-to-side Billroth I arrangement in these five patients have been gratifying. There are two features about this group of cases that are of special interest. First, although others have shown that benefit often results from converting Billroth II resections to the Billroth I type, the assessment of these beneficial results has been largely based on clinical observation alone. In this group of patients, however, the clinical improvement has been accompanied by demonstrable, objective physiologic changes which cannot be attributed merely to some psychologic adjustment by the patient. Second, a small stoma was used to modify an end-to-side, Finney-von Haberer type of gastroduodenostomy. It was believed that a small stoma gastroduodenostomy should provide maximum opportunity for delaying the

emptying time and improving nutrition. The end-to-side arrangement seemed preferable to the end-to-end because it afforded more opportunity to control stomal size. In addition, an end-to-side anastomosis is technically easier when dealing with a previously closed and perhaps buried duodenal stump.

The small stoma as described by Porter and Claman was first introduced to one of us (D. B. H.) by Harvey W. Baker [*1*] of Portland, Oregon, and is readily adaptable to either the Billroth I or Billroth II type of arrangement. While one might fear that such a lumen would obstruct easily, this has not proved to be true and the use of this small anastomosis has actually been attended by minimal difficulty. Meticulous care, however, must be used to avoid too much tissue inversion. Gentle handling of tissues is thought to be important in helping to prevent postoperative stomal edema.

All of the Billroth II resections in this group of cases were either of the Polya variety or were Hofmeister modifications with stomas of large size. The amount of resected stomach appeared to have varied between 50 per cent and 70 per cent. The patients had dumping symptoms of severe degree which were carefully defined and documented prior to reoperation by observing the results of controlled test meals and by objective physiologic changes which accompany and identify the dumping syndrome.

Vagotomy was not performed on any of these patients, either at their original operations or at the time of conversion to the Billroth I (end-to-side). There was no evidence of marginal or recurrent ulcers found in any of the cases. It seems reasonable to assume that the benefits obtained by reoperation in these patients were actually due to either the reestablishing of gastroduodenal continuity or to the effect of the small stoma. It is probable that the favorable results were due in part to both the gastroduodenostomy and the small stoma.

Cases I, III and IV are classic examples of the dumping syndrome. Each patient exhibited nausea, unpleasant warmth, varying degrees of syncope, sweating and weakness relieved only by lying down. They are representative of those dumping patients who have severe systemic symptoms probably related to unstable vascular homeostatic mechanisms [*5*]. The patient in Case II had large plasma volume changes comparable to the plasma volume changes in Cases I, III and IV, but had minimal systemic symptoms of dumping. His symptoms were largely abdominal or digestive in nature consisting chiefly of fullness, nausea and the inability to eat. The reason for the different symptomatology in these dumping patients appears to be related to differences in peripheral blood flow [*5*]. Explosive diarrhea was the chief complaint of the patient in Case V, although slight to moderate systemic symptoms such as sweating and weakness were also present. The same small stoma, end-to-side Billroth I arrangement was used in each of these patients with good results. This adds support to the concept that excessively rapid gastric emptying underlies all phases of the dumping syndrome, inasmuch as one of the chief effects of the conversion operation was to delay gastric emptying. This preliminary experience with surgical treatment of the dumping syndrome by the use of a small stoma, end-to-side gastroduodenostomy is encouraging and it suggests that dumping symptoms frequently may be controlled by this type of operative approach.

SUMMARY AND CONCLUSIONS

1. A group of five patients has been presented with severe dumping symptoms following Billroth II type gastric resections.

2. Observations were made during induced dumping attacks with regard to decreased plasma volume, changes in peripheral blood flow, electroencephalographic tracings and roentgenographic studies.

3. These patients were treated by converting the Billroth II resections to a small stoma, end-to-side Billroth I arrangement.

4. Favorable clinical results were obtained with relief from the dumping symptoms.

5. Observations with regard to changes in plasma volume, peripheral blood flow, electroencephalographic tracings and roentgenographic studies were made after reoperation which showed objective evidence of improvement.

6. This preliminary experience suggests that severe dumping symptoms may frequently be relieved by this type of surgical approach.

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DISCUSSION

Edward Woodward (Los Angeles, Calif.): Dr. Hinshaw and his co-workers have demonstrated in impressive fashion how the physiologic alterations associated with the postgastrectomy dumping syndrome can be utilized not only in its diagnosis but also in evaluation of its surgical therapy. We have performed reoperation upon fifteen patients with the dumping syndrome intent upon (1) drastically narrowing the gastroenteric stoma and (2) directing the gastric outlet into the duodenum whenever possible. The operation in ten patients has been conversion of a Billroth II type of reconstruction into an end-to-end Shoemaker-Billroth I gastroduodenostomy. The clinical results have justified the additional procedure in all cases and there has been dramatic improvement in several cases. We have been particularly interested in the changes wrought in the absorption of ingested nitrogen and fat. A nutritional balance study made in such a patient before and after conversion to a Billroth I gastroduodenal anastomosis revealed that there was no significant change in nitrogen absorption, but the fecal loss of fat was reduced from 10 per cent to 4 per cent of ingested fat. This result is comparable to that reported by other observers. This change in fat absorption is probably a rather small factor in the weight gain so commonly observed after conversion from Billroth II to Billroth I; the most important factor, it seems to us, is the ability to ingest sufficient quantities of food. The patient with the dumping syndrome is distinctly more comfortable in the fasting state and either consciously or unconsciously restricts his dietary intake toward this end.

We believe the dumping syndrome is due to the too rapid entrance of hypertonic ingested materials into the small intestine followed by the rapid pulling of intravascular fluid into the intestine. Patients with nervous and emotional instability seem to have a lower threshold and symptoms develop more readily in response to these physiologic alterations. We know of no evidence that the duodenum is more resistant to these changes than the jejunum, and it seems likely, therefore, that the dumping syndrome is less frequent following the Billroth I operation, largely because of the much smaller size of the anastomosis. Since 1947, we have been making all gastrojejunal anastomoses, with or without gastric resection, 1¼ inches in diameter and have been rewarded with a negligible incidence of the dumping syndrome. We believe that the many published reports regarding the Billroth I gastric resection indicate that it is unsafe in male patients with duodenal ulcer, unless a full three-quarter gastrectomy is performed; with a lesser resection the recurrence rate is unacceptably high. We believe this is an excessive amount of stomach to remove because of an unavoidable increase in operative mortality, as well as an excessive incidence of chronic nutritional defects. Therefore, we believe that the Billroth II reconstruction should be used when gastric resection is chosen in treating peptic ulcer, with the exceptions of benign gastric ulcer and duodenal ulcer in female patients.

I would like to ask the authors if they think prevention of the plasma volume drop by intravenous plasma and dextran will obviate the development of subjective symptoms.

CLARENCE E. STAFFORD (Los Angeles, Calif.): I would like to add two points to this discussion. One is that by these tests that Dr. Hinshaw has outlined, and previously reported, it is possible to pick up a group of what we might call "potential dumpers."

The second point is that it is hoped that as a result of these tests it may be possible to determine preoperatively in which patients dumping syndrome will develop, and then select the type of operative procedure which will tend to limit the mechanical dumping process.

The operative procedure that was demonstrated by the authors is not offered as the only solution. It merely represents an attempt to try to distinguish the various types of operative procedures which may help to correct the mechanical difficulty.

We hope that this material will lead to further investigation along this line.

JOHN RAAF (Portland, Ore.): I was very much interested in the electroencephalographic tracing that Dr. Hinshaw showed and I would like to ask a question.

The altered activity demonstrated in the segment of the electroencephalographic record lasted a few seconds and I am wondering whether there were other such "bursts" and how long they continued. Even assuming that there are hypothalamic discharges during the dumping syndrome, usually such "bursts" come from cortical lesions. The one shown here might even simulate a grand mal attack but I am sure most people with the dumping syndrome do not have epileptic seizures.

I am wondering if all five of the patients with the dumping syndrome showed the same sort of electroencephalographic tracing, and I would like to suggest that the tracing presented might be an artifact.

ROY COHN (San Francisco, Calif.): I would like to ask a question, which has always been difficult for me to understand, posed by those who advocate the narrowing of the lumen as a point in relief of the dumping syndrome.

The radiologic literature is replete with suggestions which would tend to show that the actual functioning outlet of any gastric resection is the diameter of the small bowel, so that it really does not make any difference how big we make the anastomosis. From the functional point of view this is the outlet of the stomach.

It is true you can make the anastomosis even smaller than that if you wish, but you certainly cannot make it any larger.

I think that we could interpret some of these findings in the benefits that have been described as the relief of small bowel obstruction.

The x-rays of one of these patients showed a dilated upper jejunum. It is difficult for me to understand why that should occur secondary to the size of the gastric anastomosis.

JOSEPH A. WEINBERG (Long Beach, Calif.): The most direct way to get at the problem of the so-called dumping syndrome is to inquire into the basic physiologic changes which are responsible for the disturbance and thus avoid unnatural surgical procedures which appear to induce the disturbance. The present contribution of Dr. Stafford and his associates is a contribution toward this end.

It is my impression from past experiences that the less one disturbs the normal continuity of the stomach and intestine, the less likely is the dumping syndrome to occur. It is principally for this reason that there has been a revival of interest in the Billroth I procedure in preference to the Billroth II procedure when partial gastric resection is indicated. The by-passing of ingested food away from the duodenum prevents exposure of the food to the influence of bile, pancreatic juices and the elements of duodenal secretion, and this by-passing must have a seriously disturbing effect upon digestion. It should require little argument to convince one that an operation which will retain the normal continuity and, at the same time, heal the disease should be the preferred procedure. There has been much agitation of late against the Billroth I operation because of the reportedly high incidence of recurrence of ulceration. Those who advocate the Billroth II procedure in preference to the Billroth I procedure fail to take into consideration the fact that ulceration at the site of anastomosis between the stomach and jejunum is a much more serious disease than recurrence of ulceration in the duodenum following the Billroth I procedure. To put it another way, in the case of gastrojejunal ulceration following the Billroth II procedure, the patient has only traded his former duodenal ulceration for the far more serious disease of gastrojejunal ulceration. This is a very real problem when it is considered that the incidence of gastrojejunal ulceration following the Billroth II procedure is variously estimated at 2 to 10 per cent.

DAVID B. HINSHAW (closing): In reference to Dr. Raaf's comments on the electroencephalograms, I would like to say that these were made as continuous tracings over a period of an hour or an hour and a half. The tracings were started about forty-five minutes before the attacks were induced for control purposes. Our neurology associate believed that the control tracings were normal. The dysrhythmias occurred about fifteen to twenty minutes after the test meals were given, which is the time when the dumping symptoms were manifested. The dysrhythmias persisted for several minutes during the height of the attacks. We believed that this pattern change may have resulted from decreased cerebral blood flow.

Dr. Cohn questioned the effect of the small stoma

on the emptying time of the stomach. I realize that the jejunal lumen has long been considered to be the true stomal size (as was emphasized by Reynolds, Cantor and Kennedy). Many of these stomas, however, are smaller than the jejunal lumen, and some believe that a small stoma has better sphincteric action than a large one. I think the objective findings in these cases indicate that something happened to these people, regardless of whether it was due to the change in intestinal continuity, or to the stoma, or a combination of the two.

The dilatation that is seen opposite the gastrojejunostomy is, I believe, common in patients who have the dumping syndrome. I think this jejunal bulging opposite the anastomosis has been described, particularly by Porter and Claman, as a feature of the Polya type resection.

Traumatic Retroperitoneal Rupture of the Duodenum

MURRAY L. JOHNSON, M.D., *Tacoma, Washington*

THIS subject is of general interest even though dealing with a rare condition. It is a good example of an unusual condition that is likely to occur with many types of abdominal trauma. Everyone performing abdominal surgery should be familiar with this entity; the fact that the diagnosis is frequently missed even at laparotomy makes it worth while to review the subject.

This paper will show and illustrate with reports of cases the various points of diagnosis and pitfalls into which the unwary may fall.

In 1943 when I saw my first case and reviewed the world literature, there were few reports of this entity in the literature of North America and these consisted mainly of isolated case reports. Foreign literature was somewhat more productive. This situation still prevails.

The mortality rate in published cases is dropping, and at present is 20 per cent. Schumacher [*1*] in 1910 found twenty-four cases of retroperitoneal rupture of the duodenum, with only two recoveries. Miller [*2*] added twenty-two cases through 1916, but the mortality rate remained extremely high, with only three survivals. In 1943, Johnson [*3*] found a 50 per cent mortality rate in fifty-two published cases from 1916 to 1943. In 1952, Cohn, Hawthorne and Frobese [*4*] found a 20 per cent mortality rate in twenty-five cases. In this paper, five additional cases, one resulting in death, are presented. Two of these cases were seen by the author.

CASE I. T. G. (52-4269), a thirty-seven year old workman, was admitted to Maynard Hospital, Seattle on September 9, 1952. He had been injured by an emery wheel breaking which was so explosive as to break the cast iron guard, striking the patient in the abdomen, chest and chin. The patient was in severe shock and was referred immediately to surgery, with the diagnosis of subcutaneous injury to the abdomen. Multiple transfusions of blood and plasma were given.

Dr. Bernard Mullen operated on the patient and found a complete fracture of the right rectus muscle. The liver was found to be split wide open in the area of the gallbladder. The gallbladder itself was completely broken from the liver and lay free in the peritoneal cavity with a fragment of liver. The end of the cystic duct and the common duct were not identified. The transverse mesocolon was torn but the circulation was not compromised. There was free air in the peritoneal cavity and in a search for the source, emphysema of the retroperitoneal area near the duodenum was discovered, through which bubbles were escaping.

On further exposure, a linear tear of the duodenum was found. Upon inserting a finger into the lacerated duodenum, two additional tears were discovered. These were repaired directly with silk sutures. The liver was brought together with large mattress sutures and the abdomen closed with drainage to the common duct area. Postoperatively, the patient was given intravenous aureomycin and intramuscular penicillin and banthine. Nasogastric suction was used for four days. He progressively improved and was discharged on September 30, 1952.

On May 7, 1954, the patient was readmitted to Maynard Hospital with a six month history of severe pain in the right upper quadrant of the abdomen and tenderness associated with nausea and a ten pound loss in weight.

Dr. Mullen operated upon the patient and freed up massive, dense adhesions between the liver and diaphragm and anterior stomach, duodenum and colon. Omentum was transposed up over the liver. The patient recovered nicely and has remained well since then.

CASE II. A. K. (G-8802), thirty-nine years of age, was admitted to the Vancouver (B. C.) General Hospital on September 1, 1948 with a history of having been thrown from the deck of a truck by a snapping cable, coming to rest against a hydrant, with the force of the blow on the abdomen. He

complained initially of moderate pain in the right upper part of the abdomen and right flank. He had not vomited, color was good, and he was not in shock. Examination showed a faint linear mark across the lower part of the chest and upper part of the abdomen. Tenderness was well localized to the right upper quadrant of the abdomen. The abdomen was tympanitic throughout and liver dullness was normal. The patient complained of severe pain over the upper part of the abdomen and rigidity was marked over the whole abdomen. Peristalsis was absent. The laboratory gave the following information: white blood cell count 15,000 per cu. mm.; hemoglobin, 96 per cent; urinalysis-albumin, 2 plus; red blood cells, 2 plus; white blood cells, occasional. X-ray film with the patient in the left lateral position showed "a suggestion of free gas over the right lobe of the liver."

Diagnosis of rupture of a hollow viscus was made and the patient was operated upon by Dr. A. B. Manson four and a half hours after injury. On exploration some blood was present in the supracolic compartment and right gutter; the liver showed three superficial lacerations over the right lobe; a tear was present in the gastrohepatic omentum and another at the inferior border of the foramen of Winslow. No other intra-abdominal lesions were found. Lateral to the duodenum, beneath the posterior parietal peritoneum, an edematous area was seen over which was a patchy greenish black discoloration extending upward toward the liver and outward over the kidney. In examining the area, a bubble of gas was seen to escape from the retroperitoneal tissue. An incision was made lateral to the duodenum, which was reflected, and an oblique tear 4 cm. in length was found on the lateral aspect of the second portion of the duodenum, from which escaped thick, bile-colored fluid mixed with food. The papilla gave a free flow of bile. A repair was made in two layers using chromic catgut. A large Penrose drain was brought out through a stab wound well out in the flank, and the abdomen closed. Gastric suction was employed and the patient was given fluids, electrolytes, blood and antibiotics.

Progress was favorable until the third day, when there was a copious discharge of bile-stained fluid through the stab wound. The general condition remained good. A 22F catheter with gentle suction was placed in the stab wound in an effort to collect the fistulous drainage. On the seventh postoperative day, a feeding jejunostomy was fashioned, and subsequently the entire drainage was collected and re-fed via the jejunostomy tube.

A high caloric, high protein, high vitamin jejunostomy feeding was added. The Levine tube was removed on the eleventh postoperative day. It is interesting to note that as much as 6,500 cc. of fluid was collected from the fistula for one day, with an average of 4,500 cc. of fluid for fifteen days. The largest total jejunostomy feeding for one day amounted to 8,350 cc., with an average of 6,510 cc. for fifteen days. This was given by continuous feeding.

At the end of eight weeks, the catheter was removed and one week later the discharge ceased and the jejunostomy tube was removed. Eight and one-half months and one and one-half years later, x-ray films revealed slight distortion of the second portion of the duodenum and no evidence of obstruction either at the duodenum or at the jejunostomy site.

Case III. A soldier, R. A. (14 325 735), was admitted to Madigan General Hospital on February 1, 1951. He was involved in an automobile accident on highway 99. He received a severe blow from the steering wheel of the car and was hospitalized immediately. Exploration revealed a hematoma in the mesentery, particularly at the base of the mesocolon. The retroperitoneal space was not explored. The patient was placed on Wangensteen suction and general supportive therapy postoperatively.

He appeared to be doing quite well until one week postoperatively, when the tube was removed. Within a few hours, there was a gradual onset of abdominal pain that progressed into a picture of shock by twenty-four hours. Dr. Eric Sanderson, the consultant, saw him in this condition and reexplored. The abdomen was full of air and there was a blowout of a badly contused and edematous duodenum about 2 inches below the ampulla of Vater. A resection of the duodenum and an end to end anastomosis was performed, duodenum to jejunum, anterior to the mesenteric vessels. The patient failed to rally and died in twenty-four hours.

Case IV. B. L. (77530A), a five-year old boy, was admitted to Tacoma General Hospital on July 16, 1952. He had pulled a heavy flower box over on himself. Two adults were required to lift this off and he was immediately taken to a pediatrician's office from which he was transferred to Tacoma General Hospital for observation. I saw him several hours later. Severe continuous abdominal pain and vomiting indicated that surgery was imperative. An additional significant incident in the history was that four days before this accident, he had complained of abdominal pain to a local doctor. He was hospitalized and on the basis of laboratory and physical findings an appendectomy was advised. The parents decided to defer operation however, as the boy was symptomatically improving.

The following positive findings were present when I examined the patient: He appeared acutely

ill, pulse was rapid, blood pressure normal. There were several contusions on the abdominal wall and chest, and diffuse abdominal tenderness with rebound tenderness and generalized rigidity. The white blood cell count was 20,800 per cu. mm. with 82 per cent segmented forms. The urine was negative except for 1 plus protein. Tentative diagnosis of possible ruptured intestine was made and laparotomy was performed, about eight hours after injury.

At operation, the right rectus muscle was found to be ruptured in several areas. There was about 150 cc. of old blood in the peritoneal cavity. There were many areas of dirty appearing fibrin over the intestines. The mesenteric glands were generally enlarged. The appendix was acutely inflamed and edematous. There was marked contusion of the transverse colon with a 2.3 cm. laceration of the serosa. There was contusion with hematoma and edema of the entire base of the mesentery. Hidden behind the root of the superior mesenteric artery was a practically complete contused tear of the fourth portion of the duodenum.

A "Roux Y" procedure was done after débridement, bringing the distal end of the severed bowel up to the stomach as a posterior gastroenterostomy. The end of the duodenum was anastomosed to the jejunum several centimeters beyond the gastroenterostomy. Appendectomy was then performed and the wound closed with drainage. His recovery was uneventful and he was discharged thirteen days later. When last seen at age ten, four and one-half years after injury, he showed normal development. He had no digestive disturbances. X-ray films showed a normally functioning duodenum and a non-functioning gastroenterostomy.

Case v. D. O. (2960B), a sixteen-year old boy, was admitted to Good Samaritan Hospital, Puyallup, Washington on July 17, 1953. He was kicked in the right flank by a horse. His wind was knocked out for a short period and he vomited a small amount of clear fluid about fifteen minutes later. There was persistent pain in the right side of the abdomen which caused him to double up. Pain was relieved by morphine. There was tenderness and rigidity of the right side of the abdomen, with rebound tenderness and referred rebound to the right side of the abdomen. Bowel tones were absent. Blood pressure and pulse were normal.

An x-ray film of the abdomen was negative. Blood counts were normal except for elevation of the white blood cells to 13,850 per cu. mm., with a slight shift to the left. Urinalysis showed 5 mg. per cent of albumin and 2 plus red cells.

The attending physician elected to observe the patient in the hospital, but over the next twenty-four hours increasing temperature (to 101°F.), pain and vomiting, despite nasogastric suction, indicated surgery. An intravenous pyelogram was normal. The white blood cell count rose to 18,200 per cu. mm. with further shift to the left.

At operation on July 18, approximately twenty-seven hours after injury, there was considerable reaction with induration and inflammation of the right colon and duodenal area. About 200 cc. of bile-colored fluid were present in the peritoneal cavity and there was a small pool of fluid under the peritoneum medial to the ascending colon. There was no demonstrable leakage from the duodenum or biliary tract at the time of surgery. The duodenum was not mobilized because of the induration. A cholecystostomy was performed; drains were placed in the retroperitoneal space medial to the ascending colon and lateral to the cecum. An additional subhepatic drain was placed. Penicillin and streptomycin were given and a nasogastric tube was put in place.

The following day, July 19, there was a marked vasomotor collapse, associated with an elevation in temperature to 104°F. I saw him for the first time that day. He was suffering from fluid and electrolyte deficit. The abdomen was soft and not distended. It was decided to continue the conservative course with careful watch of the fluid balance. One day later, July 20, there was a urea retention up to 56.5 associated with a persistent albuminuria and showers of hyaline and granular casts. These abnormalities subsided to normal by July 25.

Within this latter period the patient improved and remained in satisfactory condition. On July 24, slight bile-stained drainage from the retroperitoneal drain gave way to a typical duodenal fistula. On July 25, a jejunostomy with replacement of the fistulous drainage and feeding was done. Drainage from the fistula gradually lessened to insignificant amounts. X-ray films taken on August 3 showed a normal biliary tree and suggested a duodenal obstruction. On August 11, x-ray with barium revealed spasm, but no leakage from the duodenum. He was discharged on August 15 in good condition. The jejunostomy tube was removed several days later in the office of the attending physician. When last seen in January, 1957 he was perfectly well.

COMMENTS

There are a number of statements that can be made from a careful review of the literature integrated by personal experience with three cases. What must be stressed is that the mortality rate could be much lower if proper diagnosis is made. This involves, first, the decision to explore and second, the recognition of the lesion at surgery.

Retroperitoneal rupture of the duodenum is

a rare result of abdominal trauma. Therefore, our approach to the surgical indication of this condition should not be exploration with minimal signs, but rather careful observation with knowledge of the possibilities.

There are certain symptoms and signs that stand out in making the decision to operate. If after an hour or two (this may be much prolonged), there is increasing or continued abdominal pain, vomiting, particularly on taking anything by mouth, a tendency to shock, and if the abdominal signs of rigidity, rebound tenderness and absent or decreased bowel tones exist, I think any careful surgeon can make the decision to explore for a possible ruptured viscus. Accuracy of diagnosis for rupture of the retroperitoneal duodenum beyond an educated guess is possible only if other objective signs can be elicited. Emphysema of the right perirenal area demonstrated by x-ray is obviously selectively diagnostic for this area but is present only occasionally. Pus after a number of hours or red cells in the urine or pain in the right testicle may indicate lesions in the retroperitoneal area but are of course not at all specific. An intravenous pyelogram may help in evaluating possible damage to the kidney.

The recognition of the lesion at surgery is of greatest importance, for rupture of the duodenum has much too frequently been overlooked (or diagnosed only after the duodenal fistula has developed). Also, the presence of other serious lesions such as ruptured liver, kidney, pancreas or colon must not prevent one from thinking further. If at operation there is retroperitoneal hematoma, emphysema or bile staining anywhere in the duodenal area, it is mandatory to mobilize the duodenum and explore thoroughly.

TREATMENT

What one does at the time of surgery is entirely dependent upon the extent of the damage. A definitive repair is to be desired. Many procedures have been successful, from simple suture to complicated resections involving the stomach, duodenum and jejunum. One must use his own best judgment and experience in this phase of treatment, remembering that the simpler procedures are generally best. Although simple packing or draining with establishment of fistulas has resulted in ultimate cures, this cannot be considered optimum surgery and is attendant with the severe morbidity of duodenal fistulae.

Intelligent use of antibiotics, careful electrolyte and fluid balance and gastric decompression appear to be mandatory complements to surgery. The careful follow-up of well conceived, sound surgery can give a very low mortality rate to this unusual and interesting condition.

The major complications include peritonitis, retroperitoneal cellulitis, duodenal fistula and duodenal obstruction. These should be kept in mind during the operative and postoperative period in order that everything may be done to prevent their development.

SUMMARY

1. Retroperitoneal rupture of the duodenum is a rare but serious injury that may result from a variety of accidents. Its occult position makes diagnosis difficult.

2. Prompt operation, recognition and treatment of the condition during surgery can give good results even with associated severe injuries.

3. Five cases are presented, including one associated with acute appendicitis, and one associated with complete traumatic excision of the gallbladder.

Acknowledgments: I wish to thank Drs. Barney Mullen and Eric Sanderson of Seattle, Drs. A. B. Manson of Vancouver B. C., Kenneth Sturdevant of Puyallup and Madigan General Hospital, Department of the Army, for their help in gathering this information together.

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DISCUSSION

Eric R. Sanderson (Seattle, Wash.): The patient mentioned by Doctor Johnson on whom I operated, illustrates the difficulty in recognizing ruptures of the duodenum even at the time of surgery. At his initial operation, the surgeon found

a hematoma in the mesentery of the right transverse colon. This he took to be from ruptured vessels in the area and he did not expose the duodenum since he thought he had found and solved the patient's problem.

Postoperatively, on gastric suction, no air entered the duodenum and apparently bile and pancreatic juice passed along the duodenum with minimal leakage for a week. As soon as the suction was stopped, the duodenum inflated and the fat was in the fire. Large quantities of air poured into the peritoneal cavity and duodenal content along with it. Therefore, we operated with the abdomen hugely distended from intraperitoneal air and with the patient in a most precarious condition.

Closing up a hole in a badly inflamed duodenum is not easy. The longer the leakage through a ruptured duodenum, the greater the inflammatory reaction about it and the more difficult the job.

In this case, we tried to make the best of a bad situation by cutting across the duodenum below the ampulla at the junction of the second and third portions of the duodenum. This eliminated the badly torn and inflamed transverse duodenum which could not be closed by suture and which had to be removed piecemeal. The remaining duodenum was then pushed to the left behind the superior mesenteric vessels, and anastomosis between the jejunum and the second portion of the duodenum completed.

This is a technical procedure far more difficult than simple suture of a laceration recently incurred.

We were not able to do it the easy way because the points now stressed by Dr. Johnson, suspicion, early recognition, early and adequate treatment were not appreciated. Had they been, this mortality might have been avoided.

Fred J. Jarvis (Seattle, Wash.): The recognition of duodenal injury in gunshot wounds of the abdomen is undoubtedly easier than in cases of closed trauma. The presence of the penetrating wound demands celiotomy, and the track of the missile guides the surgeon to the penetrated or divided duodenum.

Cave* has reviewed the 118 wounds of the duodenum treated by the Second Auxiliary Surgical Group during World War II. It is interesting that of the nine cases seen in 1943, in the early experience, two were overlooked. As experience accumulated in 1944 and 1945 only one perforation was overlooked in 109 cases. Reflection of the right colon and of the lateral peritoneal reflection of the duodenum to expose this retroperitoneal section of the bowel became standard practice among those of us who were operating upon these men. The high mortality rate (56.8 per cent) reflected the multiplicity of wounds to adjacent pancreas, biliary tract, liver and vena cava. If the duodenum alone was injured there were no deaths.

I concur with Dr. Johnson in his opinion and even more strongly emphasize the desirability of prompt celiotomy in cases of blunt abdominal trauma as well as in penetrating wounds. Visceral damage usually exceeds prelaparotomy estimate. A policy of exploration of all abdominal cavities suspected of penetration by a missile was a major contribution to the lowered mortality rate of these wounds in our hands. Three thousand one hundred fifty-four abdominal injuries are recorded along with the 333 negative explorations of the abdomen for suspected perforation. The latter was not productive of mortality. These lessons, so painfully learned by the military surgeons, should transfer to the civilian situation. If visceral rupture is suspected, delay only long enough to accomplish resuscitation, then make the exploration complete.

Carleton Mathewson, Jr. (San Francisco, Calif.): It has been about ten years since the war and unfortunately many of the lessons that Dr. Jarvis has just re-emphasized have been forgotten. At least they are not being taught to the young men who are now being trained as surgeons.

I would like to re-emphasize what Dr. Jarvis has said: these people can be salvaged if the exploration is performed early. The mortality rate is very high if the lesion is recognized late.

I would like to make a plea to all of you to emphasize these points, because most of you have something to do with the training of young men. I am sure that most of the young men, when they go into whatever locality they practice in, are going to be the ones who are called upon to see these patients.

Gunshot wounds, stab wounds, and blunt injuries to the abdomen occur, not in the volume that they do during wartime, but they do occur. The number of automobile accidents is increasing and the number of hollow viscus injuries due to blunt blows on the abdomen is increasing because of this fact.

Once again I would like to ask all of you to emphasize to the young surgeons that they are not going to get many aortas to resect during the first five years, but that they are going to see a good many perforations of hollow viscera, and that the mortality rate is going to be very high unless they recognize them.

It is very distressing to me to find how few young men coming out of our fine institutions today know anything about trauma. I think this is a sad commentary and I would like to make a plea to all of you to re-emphasize the importance of being able to recognize intra-abdominal injuries.

I would like to repeat just one more point, and that is, in performing an exploration of the ab-

* Cave, W. H. Surgery in World War II, Vol. II, pp. 235–240. Office of Surgeon General, Department of Army, Washington, D. C. 1955.

domen, when an injury is suspected, it must be done in an orderly fashion, and the retroperitoneal portions of the bowel must not be overlooked. Injuries to the pancreas and retroperitoneal portions of the duodenum are common following blunt trauma. In order to see all portions of the abdominal cavity, a vertical incision in preference to a transverse incision is usually required. Therefore, if you are looking for trouble, make a proper incision and do a proper exploration.

HERBERT S. CHAPMAN (Stockton, Calif.): I enjoyed the essayist's paper very much.

During the twenty-seven years that I have been chief of emergency service I have performed many explorations of the abdomen. I had never observed a perforation of the duodenum until I saw two five year old girls who stumbled while running with little blocks of wood held against their abdomens; both had perforated duodenums.

MURRAY L. JOHNSON (closing): The discussers have stressed many points and I would like to review them again.

1. Careful clinical observation early in these cases of abdominal injury is certainly important, even though the injury does not seem initially to be severe.

2. If the cardinal signs are present, exploration should be made early. Here clinical judgment helps; it is usually the inexperienced doctor who does not perform an exploration early.

3. Then, of course with hematoma, bile staining, or gas, the retroperitoneal tissues should be explored; this is very easy with mobilization of the duodenum.

A Reappraisal of the Routine Use of Nasogastric Suction

DAVID H. SPRONG, JR., M.D. AND WILLIAM F. POLLOCK, M.D., *Santa Monica, California*

IN 1949 Ferguson [3] said, "The practice of preventing complications which never occur or never should occur, is so widespread that the preoperative and postoperative care of patients has become a complicated procedure for residents and interns, and more important, a most troublesome time for the patients." No better example of what he means could be selected than the routine use of nasogastric suction following ordinary uncomplicated abdominal operations. Yet it is common in hospital wards to see patient after patient wearing a gastric or intestinal tube with no indication for this other than that an operation had been performed. This, as Ferguson would say, is preventing complications that never should occur.

There can be no doubt that nasogastric suction is of great benefit and may be a life saving adjunct in the treatment of patients who have dilated bowel and stomach due to either mechanical or paralytic ileus. However, most patients do not become obstructed postoperatively, and there should be little need for intubation.

In addition to the often considerable discomfort resulting from the presence of a nasal tube, there are sometimes adverse effects or complications which may be serious in nature. Taylor [6] has said, "The nasal suction tube may become quite a dangerous postoperative tool in that it removes large quantities of gastric electrolyte. This is done insidiously without the drama and demand for action accompanying active emesis." Lichtenstein [5] lists epistaxis, sinusitis, eustachian inflammation, otitis media, laryngeal edema, esophageal erosion and stricture, gastrointestinal perforation and hemorrhage among the complications of intubation. He points out how many of these can be prevented by proper precautions.

Frank and Avola [4] studied several cases of pseudomembranous enterocolitis and found an overgrowth of Micrococcus pyogenes. They often obtained pure cultures of this organism from the tips of the nasogastric or intestinal tubes, and suggest that the infection may be introduced by these tubes. Eade, Metheny and Lundmark [1] compiled a list of 108 published cases of serious complications following intubation. "These required forty-nine immediate operations, including twenty-four tracheotomies and led to death in eight patients. Many who recovered had long convalescence and some had permanent damage."

In a study of the complications of nasogastric intubation Farris and Smith [2] sent a questionnaire to 200 members of the American Laryngological and Bronchoesophagological Societies. Of the 115 who replied fifty-nine members reported a total of 260 major laryngeal or esophageal complications; seventy-nine required tracheostomy and twenty-two required dilatation of esophageal strictures. There were several perforations of the esophagus and seven deaths.

Since intubation is both uncomfortable and dangerous it is important that its use be restricted to those situations in which it is definitely needed. If most patients undergoing a gastrectomy, a cholecystectomy, or any particular operation are going to need a tube during the early postoperative period, then its prophylactic use would be justified. If, on the other hand, it could be shown that most patients tolerate these operations well without intubation, then some more specific indications would seem necessary.

The records of eighty-three consecutive patients undergoing gastrectomy and 124 undergoing cholecystectomy were reviewed with

regard to the use of nasogastric suction during the postoperative period. Of the eighty-three patients in whom a gastrectomy was performed, twenty-six required intubation. Eleven of these had pyloric obstruction and were intubated before operation. In another patient intestinal obstruction developed on the fifth postoperative day and the patient was treated with nasogastric suction, and operation. Two patients were intubated before operation because of massive bleeding.

In the remaining cases suction was started one to six days after operation because of vomiting. The duration of the suction in this group varied from one to four days, the average being 2.2 days. An additional fifteen patients vomited small amounts during the first few postoperative days, but were not intubated. A few reported some nausea but did not vomit.

Of the 124 patients undergoing cholecystectomy only ten required intubation. Two of these patients had a perforated gallbladder with bile peritonitis. In each case suction was started shortly before operation and was continued for two days postoperatively. One patient had severe pancreatitis. Suction was used for three days preoperatively and was continued for two days after operation. Another patient with subacute cholecystitis was thought at first to have intestinal obstruction. Suction was used for one day before operation and one day after. In the other six patients a tube was passed from one to eight days postoperatively and suction continued for one to three days.

Thirty-five patients vomited small amounts during the first two days and sixteen others had some nausea. We do not consider these as adequate indications for intubation. The majority of the patients went through their postoperative convalescence with no nausea or vomiting and without benefit of tube.

Eade, Metheny and Lundmark [1] reported 143 consecutive cases of major abdominal surgery; 130 of these patients were not intubated. There were no mortalities and it was believed that there were no adverse effects from the lack of suction. The authors concluded that "intubation should be used only when specific indications exist." We agree entirely with this philosophy and consider that these specific indications are: (1) mechanical obstruction, and (2) adynamic ileus severe enough to cause distention and vomiting of significant amounts.

The occasional retching due to reflex nausea or to the narcotics used seldom warrants intubation. These symptoms can often be relieved by changing narcotics or by the use of dramamine® or thorazine.

Farris and Smith [2] have advocated temporary gastrostomy as a substitute for nasogastric intubation. This not only avoids the discomfort associated with intubation but avoids the main complications, which occur in the nasopharynx, the larynx and the esophagus. We have had no experience with this method but believe it has merit.

The function of a gastric or intestinal tube is to remove excess gas and liquid from the stomach or bowel. The mere presence of a tube exerts no beneficial effect on the patient but often seems to give the doctor a feeling of accomplishment. Frequent inspection and irrigation as needed to insure patency and an accurate record of intake and output are essential for the proper use of a tube, yet these are often neglected. The unrestricted use of fluids by mouth during nasogastric suction is dangerous but small amounts of water or other clear liquids can add greatly to the patient's comfort.

We believe that nasogastric suction is used too frequently and also that it is often continued long after the need for it is past. The decision to remove the tube is commonly based on the appearance of peristalsis. Minimal peristaltic activity will end the need for suction but will produce only fleeting and infrequent sounds, which may be missed unless considerable time is used in listening for them. The need for continued suction can best be determined by clamping the tube for a few hours and giving measured amounts of liquid by mouth. The gastric residual is then measured and the patient questioned as to discomfort during the procedure. From this test one can tell fairly accurately whether or not further suction is needed.

SUMMARY

The complications and hazards of nasogastric suction are reviewed and emphasized. A series of eighty-three gastrectomy and 124 cholecystectomy cases is presented. Intubation was needed in only 8 per cent of the cholecystectomy group and 31 per cent of the gastrectomies, almost half of whom had pyloric obstruction preoperatively. The over-all incidence in the entire series was 17.5 per cent.

It is concluded that nasogastric suction

should not be used routinely following abdominal surgery. It should be reserved for patients showing either mechanical obstruction or adynamic ileus which is severe enough to cause distention and vomiting. When suction is used it should be carefully supervised and should be discontinued as early as possible.

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DISCUSSION

Vernon O. Lundmark (Seattle, Wash.): For the past six years we have rarely used nasogastric suction in our surgical practice. We have used it only three times in 82 gastrectomies since 1951. Since that time we have used it but twice in cases involving the gallbladder.

We have been of the opinion that in most elective surgical cases postoperative vomiting or notable distention when it does occur is a dramatic event and the question is not so much suction as diagnosis. A recent case is illustrative. This patient was an elderly man who had had a subtotal gastric resection for an obstructing duodenal ulcer. He got along very well during the first four postoperative days. He had been up and about and was beginning to eat when nausea, retching and generalized abdominal distress suddenly developed. After some study a barium enema revealed a ruptured diverticulum and a small amount of barium was found in the free abdominal cavity. Certainly postoperative suction would not have prevented this complication and would have disturbed electrolyte balance and might have masked for a period the actual state of affairs.

Without question, suction is in order when distention is present preoperatively, during surgery, or postoperatively. It is logical if the surgery is an irritative one or associated with prolonged ileus, and certainly diagnostic intubation is logical. But the prophylactic use of suction is rarely warranted in the usual elective surgical procedure.

Dr. Francis Moore and others have pointed out in recent years that the physiologic response to trauma or surgery is that peristalsis shuts down normally. There is a diminution in gastric secretion and absorption which occurs from the onset of trauma for periods varying from twenty-four to ninety-six hours depending on the extent of the traumatic episode. In the usual elective case, fluid accumulation resulting in distention seems unlikely without a complication being present. When the secretory activity does return peristalsis returns to propel it along.

There are certain tricks which we have used and which we believe are helpful.

1. We use the nasogastric tube for gastric lavage in all our gastric carcinomas and in some benign ulcers particularly in the obstructive type, leaving the tube in place until after completion of surgery. This is used purely as a means of decompression. Anesthetists have a way of ballooning out a stomach occasionally with anesthetic gases.

2. We give nothing by mouth to the patient until peristalsis is very definite or even better not until after flatus is passed spontaneously.

3. Hiccoughing, nausea, and retching in other than gastric cases, may be significant. It may indicate an impending and serious gastric dilatation or it may indicate only a drug idiosyncrasy. A nasogastric tube is gently slipped down and suction applied. We believe this falls under the heading of diagnostic intubation. After lavaging out the stomach to remove regurgitated bile or if minimal secretions are present, we remove the tube immediately.

4. We start oral feeding with fluids at the rate of 1 ounce per hour and if tolerated increase rapidly. We use only fluids without sugars or carbohydrate in any form during the early postoperative period.

This eliminates a considerable amount of distention until peristalsis is quite active.

We think these expedient procedures are in keeping with known physiologic concepts.

Eugene J. Joergenson (Glendale, California): We have been conducting studies at the County Hospital in the no-tube postoperative management in abdominal surgery and have been impressed with one thing—the importance of nothing being taken by mouth during this period.

We have been able to demonstrate by x-ray studies that even the simple act of swallowing water may be associated with the ingestion of large amounts of air. Because of personnel problems, etc., rigid criteria were established to insure adequate control.

Based on these experiences, if the no-tube treatment is to be employed during the immediate postoperative period, it is recommended that nothing be taken by mouth until peristalsis is re-established.

Robertson Ward (San Francisco, California): I want to call your attention to just one word in the title of this paper, which I think justifies the

presentation of the paper and any discussion that might come from it. That is the word "routine."

I am allergic to "routine." I do not think anything should be done by routine. That is why I am glad this paper was given, because it gives me a chance to say how opposed I am to routine, whether as a word or as a procedure.

I am asked very frequently, "now, what is your routine procedure in such-and-such a case?" I always use the occasion to denounce routine.

I think that that is the object of this paper, and the lesson we should all take home with us.

If anybody is using gastric suction as a routine they should be criticized. Gastric suction is a very useful thing when necessary, and the circumstances under which it was first advocated are rapidly disappearing with the use of antibiotics, and early surgery. We are not seeing general peritonitis, and that was the case in which it was used when first devised.

I am glad that the authors presented this paper, because it gives me an opportunity to stand up and criticize "routine."

JOHN R. PAXTON (Glendale, California): I have only three points that I would like to make. First, Dr. Ward, with his usual reticence, failed to take credit for the actual clinical use of continuous nasogastric suction.

He published an article in 1926 advocating this, three years before Dr. Wangensteen, and then I think such credit should go to one of our own members here.

Secondly, regarding the use of the temporary or tube gastrostomy. This was first used, to my knowledge, ten years ago at the County Hospital by Dr. Arthur C. Pattison. While he was performing a portocaval shunt on a patient with cirrhosis and esophageal varices, he wanted to use a tube. However, because of the varices, he was afraid to insert a Levin tube; therefore, he made a temporary gastrostomy by inserting a catheter into the stomach and hooking it up to decompression. This technic has been used at the County Hospital frequently for the past ten years.

The third point that I would like to bring up is that in reporting the incidence of complications from Levin suction, we should be a little more specific; it makes considerable difference whether you use a rubber or plastic tube, and if you use a rubber tube whether it is a No. 12 or 18, etc.

Therefore, when you report complications as a result of continuous nasogastric suction, it should be included whether the tube was new or old, rubber or plastic, or whether it was a No. 16, 12 or 20, so far as size and pressure are concerned.

Dr. C. J. Berne has instituted a suggestion, so far as nasogastric suction is concerned, in that he believes at no time should a Levin tube larger than a number 14 be used.

Secondly, it is his feeling that a new tube should never be used on the basis of the stiffness which increases the irritability; he has coined the phrase that a new tube is similar to a new tight shoe; that an old boiled tube is the equivalent of a soft, well-worn shoe.

JACK M. FARRIS (Los Angeles, Calif.): I endorse the views of Dr. Sprong and Dr. Lundmark. I do think, however, a word of caution may be in order because I am convinced that when the ten most significant contributions to surgery during the twentieth century are documented decompression of the upper gastrointestinal tract in certain pathologic states will be one of those contributions.

As you well know, there are certain operations in which upper gastrointestinal decompression is mandatory such as in intestinal obstruction. Also, it is apparent that Dr. Lundmark and Dr. Sprong are not vagotomists. Otherwise they would not be able to perform eighty-two gastric operations and employ nasogastric suction only twice. I believe all who have had experience with vagotomy are of the opinion that decompression should be done in every case. We have been quite interested in the problem of complications of nasogastric suction as indicated by Dr. Sprong.

I thought you might be interested in some of the classic lesions which occur from the presence of the inlying Levin tube.* I have been aware for a good number of years that the patient not infrequently complains of a sore throat, but I was unaware of the exact nature of the pathologic cause. However, autopsy studies often show an ulceration of the uppermost attachment of the esophagus at the cricoid cartilage as a result of pressure necrosis by an inlying Levin tube positioned in the midline. If the tube is positioned in the midline for any length of time, particularly if it is large sized, there is pressure upon the mucosa of the esophagus and the interposed segment undergoes ulceration. At autopsy careful examination of an esophagus in which a Levin tube had been present for any length of time will invariably demonstrate multiple ulcerations along the course of the esophagus. If this ulcer perforates, as it sometimes does, anteriorly it results in perichrondritis of the wings of the cricoid extending into the epiglottis with edema and resulting obstruction. These are the cases in which tracheotomy is required.

The other classic lesion resulting from intubation of the gastrointestinal tract is a stricture of the esophagus which has occurred in a large number of cases and after an average of about eight days of intubation.

This lesion is most serious; some of these patients have required major reconstructive surgery in order to solve the problem.

* Dr. Farris illustrated his discussion with slides.

In this connection I believe the so-called cardiospasm sometimes seen following operations upon the upper gastrointestinal tract may be due to inflammation of the lower end of the esophagus resulting from the presence of the tube.

The third complication is perforation of the gastrointestinal tract, as has been mentioned by Dr. Sprong and Drs. Ede, Metheny and Lundmark. One perforation which occurred at the California Hospital was caused by a Mercury bag which eroded through a segment of the small bowel, producing peritonitis and death. The tube being fixed at the nose at one extremity and held in a fixed position at the other by means of the Mercury bag, peristaltic activity to and fro resulted in tamponade at one area producing an enormous ulcer along the mucosa of the stomach and esophagus and also perforation of the small intestine. This complication has also been described by many others.

As Dr. Sprong indicated, we have been interested in temporary gastrostomy as a substitute for nasogastric suction when one thinks it may be indicated for a significant period of time. Dr. Smith and I reported seventy-two instances in which we have employed this method; we now have over 150 cases in which temporary gastrostomy has been used.

A wide variety of tubes have been used with equal success although we do think a No. 18 or a No. 20 Foley bag soft rubber catheter is the one of choice.

In any operation in which you think you might want suction, for example in a vagotomy or in an intestinal obstruction which is actually present at the time of operation or following resection of an obstructed colon, and when the incision allows, we introduce a Foley bag catheter in the proximal portion of the lower half of the stomach enclosed in a single purse-string suture. The butt end of the catheter is then brought out through a stab wound at a site which is the topographic opposite of the gastrostomy site. Subsequent traction upon the tube with the balloon produces impingement of the visceral and parietal peritoneal surfaces. This fusion of the peritoneal surfaces is further insured by one or two contingency sutures. This is the safety factor in temporary gastrostomy.

Then the tubing is brought out through the stab wound and with the patient in the recumbent position the most important feature is that you now have an implement for the selective removal of air and, as you know, a patient may swallow an enormous amount of air, particularly in the immediate postoperative stage. At the same time, with the patient in the recumbent position the location of this tube ventrally placed allows fluid to traverse the pylorus in increments of physiologic tolerance. If there is temporary pyloric obstruction and this tolerance is exceeded, the tube provides an exigency vent or valve in which the fluid can be removed either by syphon or by motor suction. This selective removal of air and only fluid in amounts in excess of pyloric tolerance greatly simplifies the electrolyte and fluid maintenance of the postoperative patient.

DAVID H. SPRONG, JR. (closing): It is gratifying to have all this discussion, and I appreciate very much the remarks that have been made.

In regard to Dr. Joergenson's suggestion about giving the patient nothing by mouth, we do not quite go along with that. We believe that giving the patient small amounts of fluid orally adds a great deal to his comfort, and probably not very much to the amount of air swallowed. Even if the patient is receiving nothing by mouth, he is going to be swallowing anyway, and a good bit of that is going to be air.

I was particularly pleased to have Dr. Ward make the remarks he did regarding routines. That is one of my pet peeves also, and I think the important point we wished to bring out in this paper was that this procedure should not be used as a routine. I go along with Dr. Ward in saying that few things should be used as a routine.

Intestinal Angina

ITS SURGICAL SIGNIFICANCE

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CONSIDERABLE attention has been directed by surgeons toward acute mesenteric vascular occlusion [*1–7*]. The clinical features and the pathologic changes which occur in the gangrenous bowel wall and smaller mesenteric vessels have been well documented. Much attention has been focused on whether the primary occlusive process has been arterior or venous. Recent reports continue to stress the gravity of the disease and record a persistence of high mortality rates. Even those patients who survive extensive bowel resection have usually been problems in management.

In contrast to the acute phase, little information is available concerning the prodromal features that may be encountered in mesenteric vascular occlusion due to arterial obstruction. This prodrome, more frequent than is generally appreciated, is consistently characterized by postcibal, generalized, cramping abdominal pain that may extend to the back. The pain occurs a short time after meals and persists for one to three hours. Minimal at first, the pain steadily increases in severity as the weeks or months pass. Association of meals with this pain soon leads to a reluctance on the part of the patient to eat. Weight loss and undernutrition inevitably follow.

Physical examination, even in the presence of pain, is rather unrevealing except for evidence of weight loss and occasional mild abdominal distention. Abdominal tenderness and findings of peritoneal irritation do not occur until later. Laboratory studies and x-ray examination of the gastrointestinal tract are uniformly negative. Sedatives, oxygen, anticholinergic and vasodilating drugs produce no significant amelioration of pain. Further, no known medical measures influence the unrelenting progression of the disease.

In the same way that angina pectoris is a manifestation of intermittent myocardial ischemia, so too is this syndrome of abdominal pain a manifestation of intermittent intestinal ischemia. Whereas the increased oxygen demand in the former is produced by physical exertion, in the latter it is produced by the increased intestinal work induced by ingestion of food. In both situations the etiologic factor is the failure of diseased arteries to provide for this increased demand. Pal's crisis, intestinal claudication and abdominal angina are synonyms that have been used to label this syndrome. By definition angina signifies suffocation, whereas claudication means limping or lame. The ischemic organ responsible for the pain is the intestine rather than all of the abdominal viscera. It would thus appear that from a semantic viewpoint the term intestinal angina would most closely identify this syndrome.

Only a few of the reports on mesenteric arterial occlusion have taken cognizance of intestinal angina [*8–11*]. Its frequency as a prodrome to complete arterial mesenteric occlusion is therefore largely unknown. However, a report in which this relationship was emphasized is that of Dunphy [*12*]. He reviewed the case records of twelve patients with fatal mesenteric arterial occlusion and noted that in seven there was a history of abdominal pain which preceded the fatal episode by weeks to years. Of the four patients reported on by Benjamin [*13*] and McClenahan and Fisher [*14*], two had had intestinal angina, one for five years and the other for two years, preceding the final acute episode. It is reasonable to assume that this prodrome has frequently been present but has

been overlooked, particularly when the ill patient is seen only during the acute phase of complete arterial occlusion.

Certain pathologic findings encountered in the arteries, themselves, in mesenteric arterial occlusion have perhaps not been fully appreciated. The smaller arteries are frequently found to be collapsed and bloodless. The occluded segment usually is located at or near the origin of the superior mesenteric artery. Extension of thrombosis into the branches of this artery does not necessarily occur. Studies reveal that with the exception of rarely encountered cases of visceral angiitis, the wall of the superior mesenteric artery distal to its first few centimeters is free of disease. Carucci's [*15*] observations are of particular significance. He failed to find a single instance of atherosclerosis of the superior mesenteric artery distal to its first 2 cm. in a study of fifty necropsies performed upon patients over the age of fifty years. In all these patients there was extensive atherosclerosis of other arteries, principally the aorta, coronary or renal vessels. Thirty-three of fifty cases, however, did reveal minimal to moderate narrowing of the ostium or the first 2 cm. of the superior mesenteric artery due to atherosclerosis.

Atherosclerosis of the larger arteries seems to be primarily a disease of their bifurcations. At necropsy many aortas appear reasonably free of disease except for atherosclerotic narrowing at or near the ostia of their branches. Progressive narrowing at these sites leads finally to complete occlusion of the ostium. Survival of the anatomic part supplied by this occluded artery is then dependent on the adequacy of the collateral supply. This is equally as true for the abdominal viscera as for the upper and lower extremities. Distinct collateral anastomoses exist among the three major aortic branches supplying the gastrointestinal tract. Whereas anatomic variations exist, the major avenue of communication from the celiac axis to the superior mesenteric artery is the loop formed by the superior and inferior pancreaticoduodenal arteries. Connecting the superior and inferior mesenteric arteries is the marginal artery of Drummond. Lesser collaterals among these three vessels probably exist. Recently, arterial ligations performed upon patients with cirrhosis have demonstrated that occlusion of the origin of the celiac axis is well tolerated. Excisional surgery of abdominal aortic aneurysms has, in like manner, shown that the rich collateral permits ligation of the inferior mesenteric artery at its origin. Instances of non-fatal occlusion of the superior mesenteric artery are also recorded [*16,17*]. However, simultaneous occlusion of the superior mesenteric artery and one of the other two major aortic branches would, in all likelihood, offer a serious threat to survival.

Of the case reports on fatal mesenteric arterial occlusion preceded by intestinal angina, only two [*18,19*] were found that present sufficient necropsy information concerning the status of all three of the aortic branches to the abdominal viscera. In both of these cases the celiac axis was found to be completely occluded by atherosclerosis and a recent thrombus was found at the markedly narrowed origin of the superior mesenteric artery. The two cases to be presented herein demonstrated a similar process. This suggests that obliteration of the collateral flow from the celiac axis is necessary before narrowing of the orifice of the superior mesenteric artery will be productive of intestinal angina.

The two case reports that follow clearly depict the clinical features of intestinal angina, a prodrome to acute complete mesenteric occlusion. In the one case in which aortography was done, a positive, objective diagnosis could be made. Of equal significance has been the recognition that this syndrome is potentially curable by surgery.

CASE REPORTS

CASE 1. H. D., a forty-seven year old white man, entered St. Vincent's Hospital on October 19, 1953, for elective cholecystectomy. His complaints were cramping abdominal pain of five months' duration and weight loss for two months. The pain was located across the upper abdomen and also involved the back. It developed half an hour after meals, continued for two to three hours and then subsided. The greater the amount of food ingested, the greater would be the pain. There was no selective fat intolerance and no vomiting. Physical examination was essentially negative. Cholecystography revealed no concentration of dye within the gallbladder. An upper gastrointestinal x-ray study failed to reveal abnormalities.

On October 21st cholecystectomy and exploration of the common bile duct were performed. The gallbladder contained eight stones. Choledochostomy was negative. The patient's postoperative course seemed satisfactory until the fourth day

FIG. 1. CASE I. Photograph of necropsy findings, demonstrating ostial narrowing and thrombosis of superior mesenteric artery. The wall of the artery distal to the ostium appears essentially normal.

when abdominal distention, jaundice and fever become marked.

On October 29th abdominal exploration revealed areas of softening in the liver, a biopsy of which was interpreted as "infarction of the liver." The patient failed to improve and remained in a state of continuous mild shock with deepening jaundice. A small bowel fistula developed.

On November 4th a third operation was performed which revealed extensive gangrene of the small bowel with numerous sites of perforation. A segment of ileum was resected. Following this operation the patient remained comatose, jaundiced and febrile. Multiple intestinal fistulas developed. He died on November 13th.

Necropsy revealed extensive gangrene extending from the stomach to the splenic flexure of the colon. Multiple infarcts were present in the liver and spleen. The aorta revealed moderately severe atherosclerosis of the abdominal segment. The orifice of the celiac axis was completely obliterated. The ostium of the superior mesenteric artery was narrowed by atherosclerosis and a recent thrombus completely occluded this vessel for a distance of 4 to 5 cm. (Fig. 1.) Distal to the area of narrowing the superior mesenteric artery appeared free of atheromatous disease.

CASE II. J. F. K., a forty-six year old white man, was first admitted to the Hospital of the Good Samaritan on July 26, 1955, because of intermittent claudication of the left thigh and leg, and sexual impotency of three years' duration. Aortography demonstrated complete occlusion of the left common iliac artery. (Fig. 2.) He was discharged on July 28th.

He was readmitted on August 3rd, and on the following day endarterectomy of the distal aorta

FIG. 2. CASE II. Aortogram demonstrating complete occlusion of the left common iliac artery.

and both common, internal and external iliac vessels was performed. His postoperative course was uneventful and he was discharged on August 16th. Following surgery, claudication of the left lower extremity disappeared and sexual potency returned.

Readmission on January 15, 1956, was required for the evaluation of cramping, generalized abdominal pain of two months' duration. The pain was postcibal, coming on about fifteen minutes after eating and disappearing in one to three hours. A heavy meal was followed by more severe and more prolonged pain. The patient volunteered the information that pain would not develop if he did not eat. Physical examination revealed no abnormalities. All peripheral arterial pulsations were normal.

A diagnosis of intestinal angina was strongly suspected. Therapy consisting of oxygen and vasodilating, anticholinergic and mucosal analgesic drugs was of no benefit. Gastrointestinal x-ray studies revealed no abnormalities. Aortography with the patient in an oblique position revealed adequate filling of the superior mesenteric artery. (Fig. 3.) This finding prompted the dismissal of a diagnosis of intestinal angina. However, more alert interpretation of this film would have uncovered the correct diagnosis. Later, close inspection of the aortogram revealed an area of marked narrowing at the origin of the superior mesenteric artery. (Fig. 4.) The patient was discharged on January 24th.

The fourth admission on February 29th was for

FIG. 3. CASE 11. Aortogram taken six months after endarterectomy of the distal aorta and both common, internal and external iliac arteries. Filling of the superior mesenteric artery is demonstrated.

FIG. 4. CASE 11. Close-up view of the aortic–superior mesenteric artery junction of the aortogram in Figure 3. The India ink outline shows the markedly narrowed origin of the superior mesenteric artery.

increasing abdominal pain that now was almost constant. Physical examination revealed severe emaciation and mild abdominal distention. No abdominal masses were palpable and peristalsis was active. Mild generalized abdominal tenderness was present. Conservative therapy was instituted until March 5th, when shock developed. The patient was prepared for surgery and during induction of anesthesia cardiac arrest occurred which responded to thirty seconds of cardiac massage. Abdominal exploration was then performed with the findings of extensive, spotty, ischemic gangrene extending from the stomach to the sigmoid colon. Mesenteric arterial pulsations were absent. The mesenteric veins were collapsed and contained no thrombi. The process was too extensive for resection. The patient died five hours postoperatively.

Necropsy revealed extensive gangrene of the gastrointestinal tract involving the stomach and the small and large bowel. The liver and spleen were normal. Examination of the aorta disclosed mild atherosclerosis. The inner surfaces of the previously endarterectomized segments of aorta and iliac vessels were smooth and their lumens were patent. (Fig. 5.) The celiac axis and inferior mesenteric artery at their origins were completely obliterated by atherosclerosis. The first centimeter of the superior mesenteric artery was narrowed to a lumen of less than 1 ml. (Fig. 6.) However, distal

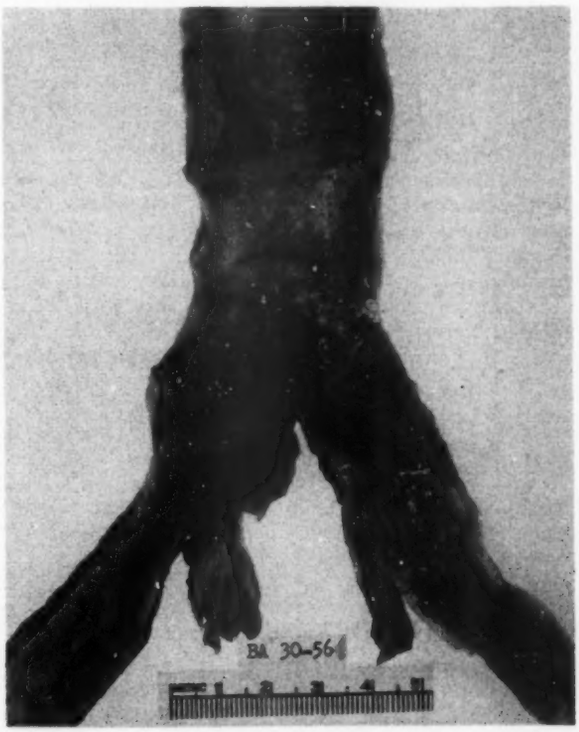

FIG. 5. CASE 11. Photograph of the necropsy specimen of the abdominal aorta and iliac arteries. The area of previous endarterectomy can be delineated.

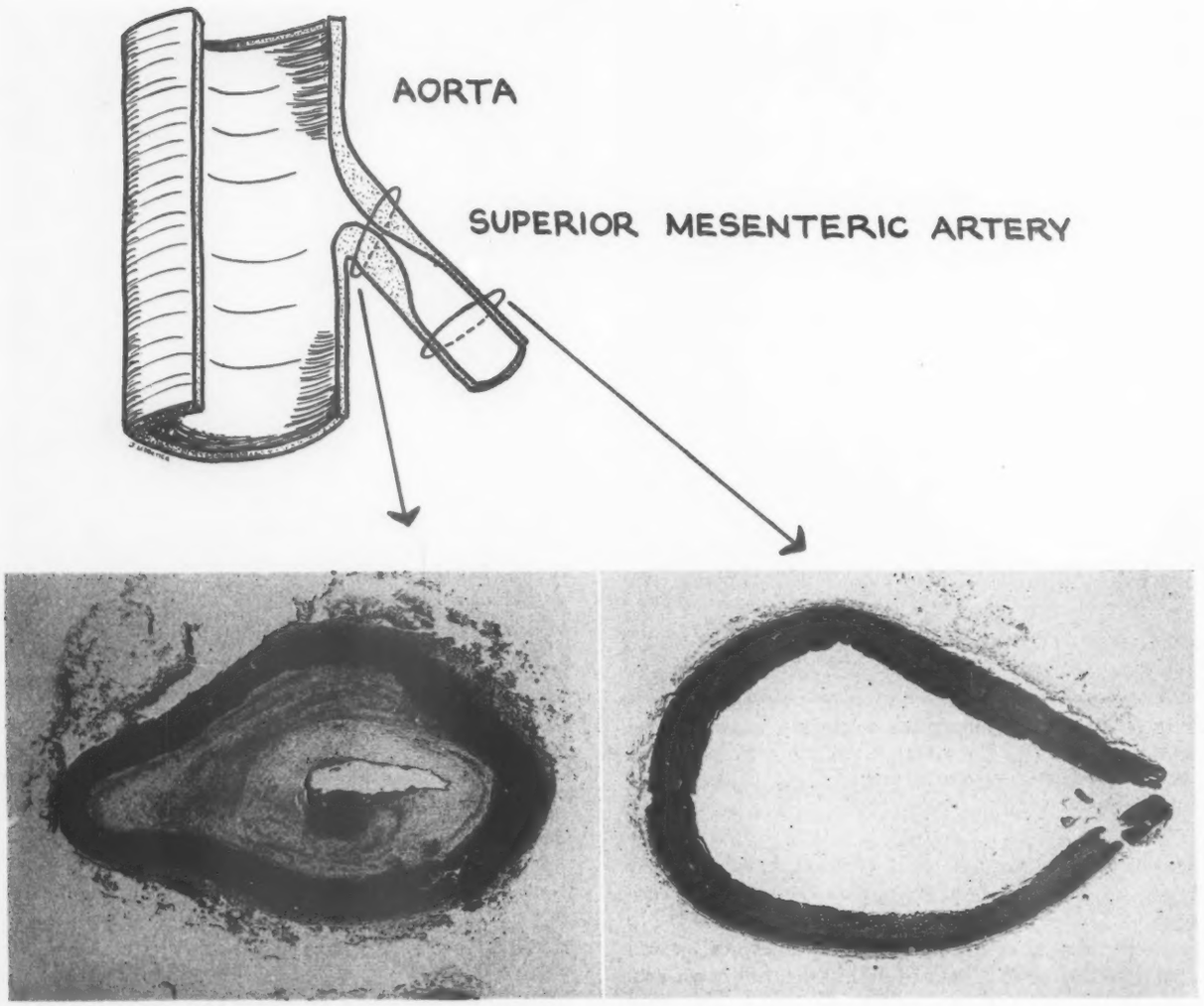

FIG. 6. CASE II. Cross section of the superior mesenteric artery taken from the areas indicated. Almost complete obliteration by atherosclerosis of the first portion is compared to the essentially normal appearance of this artery 2 cm. distally.

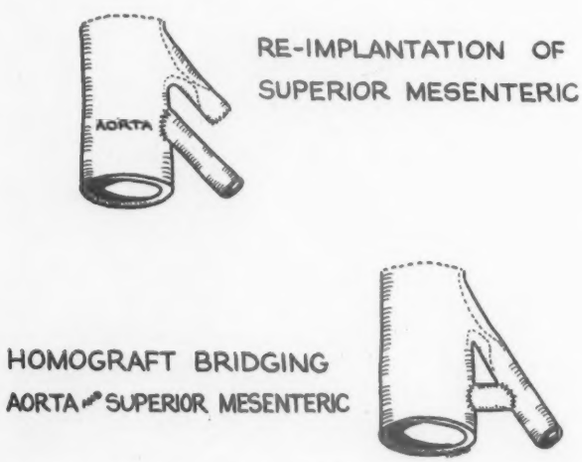

FIG. 7. Diagrams of potential surgical procedures designed to circumvent the area of narrowing at the origin of the superior mesenteric artery.

to this area of narrowing the artery was normal. There was no thrombus in this vessel.

COMMENTS

The classic symptoms and findings encountered in intestinal angina are exemplified by these two case reports. The correct diagnosis was completely overlooked in the first case, and although strongly suspected in the second case, was abandoned when the aortogram was incorrectly interpreted. It was not until the necropsy findings prompted a "second look" at the aortogram that the lesion was delineated. The important contributions of these cases have been that the character of the pain in this syndrome is almost sufficient by itself to permit a diagnosis to be made and that filling of the

superior mesenteric artery when seen on aortography does not eliminate it. Perhaps in other cases, as in the second case herein, a narrowed orifice of the superior mesenteric artery will be consistently visualized and allow a positive diagnosis to be made on objective grounds.

The process in each of these patients was one of steadily increasing pain and unrelenting progression of disease to the final stage of intestinal gangrene. Conservative or expectant therapeutic measures were of no benefit in preventing a continuing diminution of intestinal blood flow.

Inspection of the necropsy specimens indicates that surgical correction is technically feasible. Restoration of adequate intestinal blood flow is permitted by the rather large caliber of the superior mesenteric artery, the diameter of which averages 9 mm. [20]. It will be recalled that this vessel immediately distal to its origin is usually normal. Because of the anatomic location of the origin of the superior mesenteric artery, endarterectomy by present available methods would be difficult. More likely procedures to circumvent the area of obstruction would appear to be transection with reimplantation of the superior mesenteric artery into the aorta, or bridging this vessel and the aorta by means of a short bypass homograft. (Fig. 7.)

Cadaver dissection has demonstrated that either of these two procedures are technically feasible by mobilization superiorly of the third and fourth portions of the duodenum, pancreas and the left renal vein. Approximately 3 to 4 cm. of the superior mesenteric artery proximal to its first branch are thus exposed. The aorta lies in close proximity directly posteriorly.

SUMMARY

The term intestinal angina most closely identifies the syndrome that may, for a period of months to years, precede complete mesenteric arterial occlusion. This syndrome is characterized by postcibal, cramping abdominal pain and is produced by intestinal ischemia. The frequency with which this syndrome occurs and its pathophysiology are discussed. Evidence is presented suggesting that complete occlusion of the celiac axis must be present before narrowing of the ostium of the superior mesenteric artery will be productive of intestinal angina. Two typical case reports are presented. In one patient an objective diagnosis could be made by means of aortography. Of noteworthy significance has been the observation that whereas medical measures were of no benefit in preventing the unrelenting progression of this disease, the potential for surgical correction does exist. The potential surgical technics are indicated.

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DISCUSSION

Jack A. Cannon (Los Angeles, Calif.): For rather obvious reasons I do not have any slides that would illustrate any point of discussion, and

Dr. Mikkelsen was kind enough to let me refer to two of his to open this discussion, which I would like to do in order to attempt to illustrate a point regarding aortograms.

If you will remember, in his presentation (Fig. 2), Dr. Mikkelsen mentioned that there was filling of the celiac axis and of the splenic artery in the original aortogram. Because the patient is in a true anteroposterior position, we cannot say too much about the inferior and superior mesenteric arteries except that there is some filling there. Then when in order to show this superior mesentery the repeat aortogram was taken with the patient in a properly oblique position, we find very poor filling of the celiac axis, as emphasized by poor filling in the hepatic arteries. However, I believe if we look very closely, even on Figure 3 it is possible for us to see the definite narrowing in the superior mesenteric artery just below its origin from the aorta.

The point I wish to make is that Dr. Mikkelsen made the definite diagnosis on clinical grounds with regard to the cause of this patient's trouble, and he made it at a time when an operation would have had a chance to cure the patient. He even had the operation planned. He then did the aortogram and was not able to interpret the aortogram in a confirmatory fashion, and unfortunately neither was the roentgenologist.

It seems obvious that obliterative arteriosclerosis is becoming increasingly a surgical disease. I think we will agree that operation on the terminal aorta and its distal branches is now an accepted part of our surgical armamentarium. Obstructive lesions of the internal carotid and renal arteries due to arteriosclerosis are being attacked with increasing frequency. There is no doubt that Dr. Mikkelsen's provocative paper will help lead the way to successful attacks on lesions in the superior mesenteric and celiac arteries in the future. Dr. Mikkelsen has obviously proved that early recognition is possible, and actually that is the only necessary requirement in this situation. We do not even have to have the aortogram. I am also confident that selected cases of coronary atherosclerosis may become amenable to surgical attack in the near future. All these lesions are similar. They attack arterial bifurcations, are segmental, and early involve relatively large arteries where the distal tree, at least initially, is patent. When a thrombus occurs, it is always a secondary manifestation.

An observation which is perhaps worth remembering and emphasizing is that any operation for arteriosclerosis must be considered palliative. It is going to be palliative until a method of preventing the relentless progression of the problem in a generalized fashion is somehow arrived at. At present it is very obvious that there is no medicine which is effective in the treatment of arteriosclerosis. Dr. Beck has mentioned many times that you simply cannot treat a patient with coronary artery disease with an electrocardiogram.

I have felt, from my own experience with a fair number of people with obstructive arteriosclerosis, that diet would seem to be increasingly important in the etiology of this condition. There are many examples which can lead one to this conclusion, and in general these examples come around to the point that populations which are underfed, or at least populations which have a low intake of fat, do not tend to show arteriosclerosis. During the war in Europe the incidence of arteriosclerosis or atherosclerotic disease went down with the caloric and fat intake, and it has gone up again with the rise of caloric and fat intake. Consequently I feel obligated to say something to a postoperative patient in this regard. I tell him in an adamant way that I think it is of extreme importance for him to reduce down to the low side of his thinnest adult weight. I also tell him to avoid fat and excess calories from then on. If you question these patients with regard to their weight when they were twenty years old, invariably you will find that they have gained 20 to 30 pounds.

I think you will agree that all of us have undoubtedly seen thin, wispy ninety year old people. We have to stop to think a minute to remember very many obese ninety year oldsters.

Willard E. Goodwin (Los Angeles, Calif.): We, as urologists, use arteriography a great deal. I should like to add some remarks about technic which may be helpful in studying suspected cases of this disease.

About half the arteriograms we take are done by the transfemoral method, i.e., needle puncture of the femoral artery, followed by passage of a polyethylene tube through the needle to any desired level in the aorta.*

This allows the arteriogram to be made with the patient lying on his back or in an oblique position. As well, the intra-aortic injection may be placed at any level the operator desires.

If you wished to study such a patient arteriographically, this transfemoral technic could be employed in order to find the right level. Multiple small injections at different levels can be made without fear of multiple aortic punctures as in the translumbar technic.

Alfred A. Strauss (Palm Springs, Calif.): I am very much interested in Dr. Mikkelsen's article on intestinal angina. We have had two cases with similar symptoms in which the patient had three attacks within a period of five years. The most striking thing about the patient was the sudden onset of pain with marked local tenderness in the

* Walter, R. C. and Goodwin, W. E. Aortography and retroperitoneal oxygen in urologic diagnosis: a comparison of translumbar and percutaneous femoral methods of aortography. *J. Urol.*, 70: 526–537, 1953.

left upper quadrant of the abdomen, with a certain amount of restlessness and anxiety. The tentative diagnosis was volvulus or mesenteric thrombosis. The indication was quite clear to perform an exploratory operation. About 12 inches of small bowel were thickened, edematous, red and meat-like in appearance. The blood supply above and below the lesion seemed quite definite, so a resection and end-to-end anastomosis were performed. The patient made an uneventful recovery.

After this operation the patient was warned that if he had a similar attack he must not wait but go immediately to the hospital or he would be apt to lose his life. About fourteen months later, while driving from or to his office, he again had a severe attack in his abdomen and immediately drove to the hospital. Again the symptoms were similar with the same clinical findings. This time about 18 inches of bowel had to be resected. The patient made an uneventful recovery.

About three years later the patient again had a similar attack and again went to the hospital. This time about 12 inches of bowel were resected about 4 feet above the ileocecal valve. The patient made an uneventful recovery.

This patient has been carefully studied as to heart and other sources which might cause this condition. In studying the blood vessels of the dissected portion, it seemed that the vessels were somewhat narrowed and had, on cross section, an increased amount of connective tissue as compared with normal blood vessels of the mesenteric vessels.

The second case was very much like the first one. Between the first and second operations there was a period of three years. The pathologic findings were about the same. The first patient's last operation was performed three years ago and he seems well; it has been a year and a half since the second patient's operation. The arteriogram taken on both patients found no cause for this condition. I think the description given by Dr. Mikkelsen of this clinical condition is very interesting because very few men so far have found by arteriogram the real cause of this condition.

WILLIAM P. MIKKELSEN (closing): I wish to thank Drs. Cannon, Goodwin and Strauss for their comments.

Dr. Goodwin's approach to arteriography is well taken. Perhaps the best comment I can make about it is that if ever I am fortunate enough to see one of these patients again, I believe that I shall forego aortography.

Acute Diverticulitis of the Cecum Encountered at Emergency Surgery

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SINCE this condition is almost always found unexpectedly at emergency abdominal operation, an understanding of it is of general interest. After an initial encounter with it, we have collected fourteen new cases, reviewed the recent literature and wish to emphasize two practical conclusions: (1) there is a hidden type of diverticulitis of the cecum and ascending colon which produces a mass indistinguishable from carcinoma until the specimen is examined by the pathologist, and (2) immediate resection for this type of diverticulitis appears to be a wise procedure under certain conditions.

Material. Among 6,781 emergency abdominal operations performed from 1948 to 1956 at the Los Angeles County General Hospital, fourteen cases of proved acute diverticulitis of the cecum were encountered. A gross pathologic description and microscopic identification was obtained in ten cases; in three the surgeon's description of the diverticulum was accepted, and proof of existence of the diverticulum was obtained in one patient from a barium enema at a later date. This is the largest single series yet reported.

An extensive search of the world literature of the last five years was made, since the review by Lauridsen and Ross [*28*], and ninety-four acceptable new cases were tabulated. (Tables I and II.)

Incidence and Symptomatology. No previous report was found in the literature giving the occurrence of acute cecal diverticulitis per emergency abdominal operation. The present study indicates that the surgeon can expect to encounter it once in about 500 celiotomies performed for an acute condition of the abdomen. (Table III.) The hidden type, in which the differential diagnosis between this and carcinoma could not be made safely until resection was performed, was met with once in 1,100 operations. The more usual type, in which a projecting diverticulum is recognizable as such, occurred once in 800 operations. The preoperative diagnosis in the fourteen reported patients with acute cecal diverticulitis was: acute appendicitis, ten; intestinal obstruction, three; and perforated ulcer, one.

A review of the literature indicates that the first case was reported in 1912 [*41*]. Other reports followed [*3*], and in 1947 Anderson [*2*] was able to collect only ninety-six cases from the world literature up to that time. He added eight more from the files of the Mayo Clinic. By 1952, Lauridsen and Ross [*28*] could locate a total of 149 cases and added four of their own. We have been able to review an additional 106 cases reported by forty-two authors appearing in the literature of the last five years, and are adding fourteen new cases, making a grand total of 273 available for study. Among the 106 recently reported cases, ninety-four (Tables I and II) were encountered at emergency abdominal surgery and are the subject of special analysis in this paper. Additional statistical information gleaned from this review is listed in the tables.

Tabulation of the preoperative diagnoses made in the recent literature are recorded in Tables I and II. Among ninety-four cases, 83 per cent were acute appendicitis, 5 per cent appendiceal abscess, 4 per cent diverticulitis, 4 per cent carcinoma of the cecum, 2 per cent obstruction and 1 per cent cholecystitis. These percentages are a good indication of the signs and symptoms which these patients present.

TABLE I

EMERGENCY ABDOMINAL SURGERY WITH A MASS IN THE CECUM DIAGNOSED AS CARCINOMA, SUBSEQUENTLY PROVED TO BE DIVERTICULITIS*

Author and Year Reported	Patient's Age (yr.) and Sex	Preoperative Diagnosis	Size of Mass	Location	Procedure Performed	Result	Size of Ostium (cm.)	Length of Diverticulum (cm.)
Cangelosi and Khedroo, 1950 [8]	55, F	Acute appendicitis	4 cm.	Anterior	Exteriorization	Well	...	2
Heikel, 1950 [21]	62, F	Carcinoma	1 cm.	Anterior	Right colectomy	Well	...	..
Kirkman, 1950 [27]	25, F	Acute appendicitis	5 cm.	Anterior	Right colectomy	Well	...	..
Mino and Livingstone, 1950 [34]	35, M	Acute appendicitis	3 cm.	Anterior	Right colectomy	Well	1.5	..
Newton, 1950 [35]	41, F	Acute appendicitis	.....	.....	Right colectomy	Well	...	..
Sartor, 1950 [46]	37, F	Acute appendicitis	1 cm.	Anterior	Right colectomy	Well	...‡	..
	32, F	Acute appendicitis	1.5 in.	Anterior	Local excision	Well	...‡	..
Tasker, 1950 [50]	64, M	Acute appendicitis	2 cm.	Posterior	Right colectomy	Dead	...	..
Gandy and James, 1951 [17]	62, M	Acute appendicitis	5 cm.	Lateral	Right colectomy	Well	...	..
Reid, 1951 [43]	30, F	Acute appendicitis	6 cm.	Posterior	Right colectomy	Well	...‡	..
Smith, 1951 [48]	29, M	Acute appendicitis	2 cm.	Anterior	Right colectomy	Well	.5‡	1
Vaughn and Narsete, 1952 [56]	.. F	Acute appendicitis	6 cm.	Anterior	Right colectomy	Well	...	..
Bogetti, 1953 [4]	34, M	Acute appendicitis	3 cm.	Posterior	Right colectomy	Well	...	..
Collins, 1953 [10]	42, M	Acute appendicitis	3 cm.	Medial	Right colectomy	Well	.5	..
	72, F	Acute cholecystitis	4 cm.	Posterior	Right colectomy†	Well	.8‡	..
Duben, 1953 [13]	32, F	Acute appendicitis	3 cm.	Anterior	Right colectomy	Well	.5	2
Geist, 1953 [18]	48, F	Acute appendicitis	.....	.....	Transverse colostomy§	Well	...	..
Unger, 1953 [52]	56, M	Acute appendicitis	3 cm.	Lateral	Right colectomy	Well	...	..
Butler and Miller, 1954 [7]	32, M	Appendiceal abscess	1 cm.	Lateral	Right colectomy	Well	...	1
Sanderson and Madigan, 1954 [44]	42, M	Acute appendicitis	2.5 in.	Posterior	Right colectomy	Well	...	..
Waite, 1954 [57]	47, M	(?) Carcinoma	.....	Posterior	Right colectomy	Well	...	..
	43, M	(?) Appendicitis	.....	Posterior	Right colectomy	Well	...	..
	38, M	Appendicitis	.....	Medial	Right colectomy	Well	...	..
	43, F	Acute appendicitis	.....	Posterior	Cecectomy	Well	...	..
	32, M	(?) Carcinoma	6 cm.	Posterior	Cecectomy	Well	...	..
Boozer, 1955 [5]	45, F	(?) Carcinoma	6 cm.	Anterior	Right colectomy	Well	...	..
Dexter, 1955 [12]	28, F	Acute appendicitis	5 cm.	Posterior	Right colectomy	Well	2	..
Sarni, 1955 [45]	29, F	Acute appendicitis	4 cm.	Posterior	Right colectomy	Well	.7	1
Updegrove, 1955 [53]	55, M	Acute appendicitis	7 cm.	Lateral	Right colectomy	Well	...	2
	48, F	Acute appendicitis	2 cm.	Posterior	Right colectomy	Well	...	..

* The average age of this group of patients was forty-two years; fourteen were males and sixteen were females. The size of the ostium and length of the diverticulum was not given in twenty of the cases and is assumed to be very small or microscopic. The average size of the recorded ostium was 1 cm. in diameter, ranging from .5 to 2 cm. The length averaged 1.5 cm., ranging from 1 to 2 cm. An extensive surgical procedure was performed in all: primary right colectomy in twenty-three, a staged procedure in three, and cecectomy in two. The average size of the mass at surgery was 3.7 cm. The position was posterior in twelve, lateral in four, anterior in nine, and medial in two. The preoperative diagnosis was acute appendicitis in twenty-five, carcinoma of the cecum in four, and cholecystitis in one.

† Indicates two-stage procedure.

‡ Indicates the presence of muscle fibers in the pathologic specimen.

§ Indicates later exploration and no lesion found.

THE HIDDEN DIVERTICULUM

This paper deals especially with a variety of acute diverticulitis of the cecum and right colon which produces a mass within the bowel wall (Cases I to VI). (Table IV.) The diverticulum may be only 1 or 2 mm. in diameter with its entire length within the wall of the bowel (Fig. 1), but not extending as far as the outer surface of the serosa. The major inflammatory reaction is thus in the serosal layer (Fig. 2), as indicated in the cross section of the mass in Case I in this series. This produces a firm, fairly circumscribed mass from 2 to 6 cm. in diameter and thickness. (Fig. 3.) There is no diverticulum on the surface nor can one be demonstrated by superficial dissection. It can be revealed only by careful gross inspection of the mucosal surface beneath the mass. The pathologist may fail to see the tiny opening and it will then be revealed only if the section transects it (Fig. 4, Case II). Another variety of the "hidden" type is produced by larger (.5 to 2 cm.) pockets in the bowel wall which extend from the mucosal side to, but not beyond, the serosa (Cases V and VI). From a study of the reports in the literature (Table I), thirty cases appear to fall into this group. In none could a diverticulum be identified at surgery. In all but two, resection of the colon was performed and the diagnosis made by the pathologist. In sixteen of these thirty cases, although the pathologist identified a diverticulum, its size was not mentioned

TABLE II

CASES CONSIDERED ACUTE DIVERTICULITIS AT EMERGENCY SURGERY*

Author and Year Reported	Patient's Age (yr.) and Sex	Preoperative Diagnosis	Identified by:		Site	Procedure Performed	Pathologic Confirmation	Result
			Appearance†	Other‡				
Kazmierski, 1950 [24]	37, F	Adhesions	(?)Yes	.....	Anterior	Local excision	Yes	Well
Newton, 1950 [35]	54, M	Acute appendicitis	.....	F	.....	Nothing	.....	Well
	.. M	Acute appendicitis	.....	.....	.....	Drain	Yes	Well
	.. M	Acute appendicitis	.....	.....	.....	Exteriorization	.....	Well
	.. M	Acute appendicitis	.....	F	.....	Inversion	.....	(?)Well
	.. M	Acute appendicitis	Yes	F	.....	Local excision	.....	(?)Well
	44, M	Acute appendicitis	Yes	.....	.....	Local excision	Yes	(?)Well
Shaw and Siegler, 1950 [47]	42, F	Acute appendicitis	.....	D	Posterior	Local excision	Yes§	Well
Gandy and James, 1951 [17]	28, F	Acute appendicitis	Yes	F	Anterior	Local excision	Yes	Well
Gilfillan, 1951 [19]	44, M	Acute appendicitis	.....	F and O	Posterior	Local excision	Yes§	Well
Reid, 1951 [43]	70, F	Appendiceal abscess	Yes	.....	Anterolateral	Inversion	.....	Well
	20, M	Acute appendicitis	Yes	F	Anterior	Local excision	Yes§	Well
Van Meter and Floyd, 1951 [54]	21, F	Acute obstruction	Yes	F	Anterior	Inversion	.....	Well
Ahlquist and Kepl, 1952 [1]	21, F	Acute appendicitis	Yes	.....	Anterior	Local excision	Yes	Well
Churney and Wilson, 1952 [9]	23, M	Acute appendicitis	Yes	O	Anterolateral	Local excision	Yes	Well
Ellis and Windham, 1952 [14]	57, F	Acute appendicitis	Yes	.....	Anterior	Local excision	Yes§	Well
Forni, 1952 [16]	63, M	Acute appendicitis	Yes	F	.....	Local excision	Yes§	Well
Jemerin, 1952 [23]	53, M	Diverticulitis	Yes	F	Anteromedial	Local excision	Yes§	Well
Vaughn and Narsete, 1952 [56]	.. M	Acute appendicitis	Yes	.....	Anterolateral	Inversion	.....	Well
	.. M	Acute appendicitis	Yes	.....	Anterolateral	Local excision	Yes	Well
	.. F	Appendiceal abscess	Yes	.....	Anterolateral	Local excision	Yes	Well
Bogetti, 1953 [4]	45, F	Acute appendicitis	.....	F	Anterolateral	Local excision	Yes	Well
	24, M	Acute appendicitis	Yes	.....	Anterolateral	Local excision	Yes	Well
	43, F	Acute appendicitis	Yes	.....	.....	Local excision	Yes	Well
	47, M	Acute appendicitis	Yes	.....	Anterolateral	Local excision	Yes§	Well
	30, M	Acute appendicitis	Yes	.....	Anterior	Local excision	.....	Well
Collins, 1953 [10]	31, M	Acute appendicitis	Yes	.....	Anteromedial	Drain	.....	Well
	38, F	Acute appendicitis	.....	C	Anteromedial	Cecotomy	.....	Well
	50, F	Carcinoma	.....	D and B	Posterior	Drain	Yes	Well
Geist, 1953 [18]	33, M	Acute appendicitis	Yes	.....	Anterior	Local excision	Yes	Well
	7, F	Acute appendicitis	Yes	.....	Anterior	Excision, drain	Yes	Well
Kirk, 1953 [26]	31, M	Acute appendicitis	.....	O	Anterior	Local excision	Yes	Well
	19, M	Acute appendicitis	.....	.....	.....	Cecotomy	.....	Pain
Unger, 1953 [52]	60, F	Acute appendicitis	Yes	.....	Anterolateral	Inversion, drain	Yes	Dead‖
	64, M	Appendiceal abscess	.....	.....	.....	Right colectomy	Yes	Well
	32, M	Acute appendicitis	Yes	B	Anterior	Drain	Yes	Well
	28, M	Acute appendicitis	.....	O	.....	Nothing	.....	Well
	52, M	Appendiceal abscess	Yes	.....	Anterior	Local excision	Yes	Well
Pelner and Puderbach, 1954 [39]	30, M	Acute appendicitis	Yes	F	Anterolateral	Local excision	Yes	Well
Waite, 1954 [57]	36, M	Acute appendicitis	Yes	.....	Anterior	Appendectomy	.....	Well
	23, F	Acute appendicitis	Yes	F	Anterior	Appendectomy	.....	Well
	51, F	Diverticulitis	.....	.....	Anterior	Right colectomy	Yes	Well
	38, M	Diverticulitis	.....	.....	.....	Right colectomy	Yes	Well
	17, M	Acute appendicitis	Yes	.....	Anterior	Local excision	Yes	Well
	26, M	Acute appendicitis	Yes	.....	.....	Local excision	Yes	Well
	55, M	Acute appendicitis	.....	B	Anterior	Suture	Yes	Well
Yovanovich, 1954 [60]	32, M	Acute appendicitis	(?)Yes	B	Posterior	Drain	Yes	Well
	25, M	Acute appendicitis	(?)Yes	B	Anterior	Drain	Yes	Well
	34, M	Acute appendicitis	(?)Yes	B	Anterior	Cecotomy	Yes	Well
	46, M	Acute appendicitis	(?)Yes	B	Anterior	Cecotomy, drain	Yes	Dead‖
Boozer, 1955 [5]	34, F	Acute appendicitis	Yes	.....	Anterolateral	Local excision	Yes	Well
	18, M	Acute appendicitis	Yes	.....	Anterolateral	Insert drain	Yes	Well
	36, M	Acute appendicitis	.....	C	Anterolateral	Inversion	.....	Well
	53, M	Acute appendicitis	.....	F	Posterior	Drain	.....	Well
	45, M	Acute appendicitis	Yes	.....	Anterior	Excision, drain	Yes	Well
	39, M	Acute appendicitis	.....	F	Anterolateral	Drainage	.....	Well
Leichtling, 1955 [29]	46, M	Acute appendicitis	.....	.....	Anterior	Drainage	.....	Well
Nissenbaum et al., 1955 [36]	59, M	Diverticulitis	Yes	F	Anteromedial	Local excision	Yes§	Well
Puestow and Wheeler, 1955 [42]	.....	Acute appendicitis	.....	F	.....	.....	.....	.....
	.....	Acute appendicitis	.....	F	.....	.....	.....	.....
	.....	Acute appendicitis	.....	?	.....	.....	.....	.....
Updegrove, 1955 [53]	57, M	Acute appendicitis	.....	C	Anterior	Drain	.....	Well
	48, M	Acute appendicitis	.....	C and O	.....	Drain	.....	Well
	72, M	Acute appendicitis	Yes	.....	Anterior	Local excision	Yes	Well

* In these sixty-four cases the age was stated in fifty-four instances and averaged forty years. There were forty-four males and seventeen females.

† Under "Appearance," when the surgeon identified a diverticulum by observing a tiny, finger tip–like process or superficial nodule, "Yes" is recorded (thirty-nine cases).

‡ Under "Other," the capital letters have the following meaning: F indicates identification of the diverticulum by presence of a palpable fecalith (seventeen cases), D indicates revealing the diverticulum by dissection from the surrounding fat and serosa (four cases), O by opening the cecal wall at a distance from the lesion and identifying the ostium (four cases), C indicates local cecitis or inflammation of the cecal wall without a mass (four cases) and B, the diagnosis was confirmed by direct biopsy (seven cases).

§ Indicates the presence of muscle fibers in the microscopic section (nine cases).

‖ Treated before antibiotics were available.

and it is presumed to have been found on microscopic section and was similar to that in Cases I to IV in this series.

HIDDEN TYPE OF CECAL DIVERTICULITIS WITH MASS

CASE I. A. D. (Hosp. No. 1520-990), a forty-three year old woman, was admitted on October 5, 1954, with right lower quadrant pain of three weeks' duration. There had been vomiting and increasing pain for six days. Previous history was negative. Her temperature was 97.6°F., pulse 88. The abdomen was soft with tenderness in the right lower quadrant. White blood cell count was 10,000 per cu. mm. The preoperative diagnosis was acute appendicitis.

At surgery a normal appendix was visualized. In the adjacent cecum a 3 by 2 cm. mass was seen and felt. (Fig. 3.) The serosa over the mass was smooth, although slightly injected. Palpation through the opposing wall of the cecum revealed it to be quite firm and apparently entirely within the wall as no intraluminal projection could be felt and no fecalith identified. Carcinoma was considered likely. Neomycin, 1 gm. in 60 cc. of saline, was injected into the ileum. Both the appendix and mass could be excised by clamping the cecum just distal to the ileocecal valve. Right colectomy was contemplated if malignancy were revealed.

Upon opening the specimen, a tiny orifice measuring 2 mm. in diameter entered a mucosal-lined cavity measuring 5 mm. in depth. Section through

TABLE III
INCIDENCE OF ACUTE CECAL DIVERTICULITIS PER EMERGENCY ABDOMINAL EXPLORATION

Preoperative Diagnosis	No. of Cases	No. of Operations	Incidence
Hidden Type of Diverticulitis			
Acute appendicitis	4	4,064	1:1,000
Intestinal obstruction	2	1,621	1:800
Perforated ulcer	..	1,095	.....
Total	6	6,780	1:1,100
Recognizable Type of Diverticulitis			
Acute appendicitis	6	4,064	1:800
Intestinal obstruction	1	1,621	1:1,600
Perforated ulcer	1	1,095	1:800
Total	8	6,780	1:800
Total for both types of diverticulitis	14	6,780	1:500

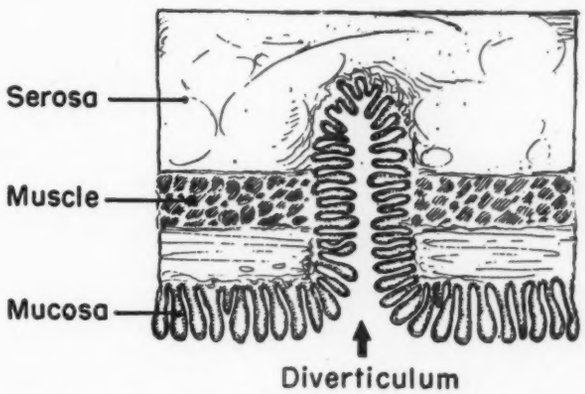

FIG. 1. Cross section of postulated microscopic diverticulum (contained within cecal wall). Infolding of the mucosa has penetrated the muscle layer and extended into but not through the serosal layer. This is probably of embryonic origin, and when the diverticulum becomes infected produces the hidden form of the disease. × 24. (Modified from MAXIMOW, A. A. and BLOOM, W. A Textbook of Histology, 5th ed. Philadelphia, 1948. W. B. Saunders Co.)

TABLE IV
HIDDEN CECAL DIVERTICULUM ENCOUNTERED AT EMERGENCY SURGERY FOR AN ACUTE CONDITION OF THE ABDOMEN

Case	Description	Site	Procedure Performed	Course	Result
I	3 by 2 cm. mass with mild inflammation	Anterior	Cecectomy	Uneventful	Well 7 mo.
II	2 cm. hard tumor	Lateral	Right colectomy	Uneventful	Well 1 mo.
III	6 by 8 cm. mass above cecum	Anterior	Right colectomy	Unsteady	Well 3 yr.
IV	Mass extends over cecum and above	Anterior	Ileocolostomy and right colectomy	Uneventful	Well 2 yr.
V	Thickened cecal wall with tiny perforation	Anterior	Right colectomy	Uneventful	Well 6 mo.
VI	Thickened hard area, ascending colon	Circumferential	Right colectomy	Uneventful	Well 1 yr.

the mass and tiny pouch revealed it to be a diverticulum. Microscopic sections showed mucosa but no muscle layer. A seven-month follow-up revealed no further gastrointestinal symptoms.

CASE II. T. S. (Hosp. No. 1582-660), a forty-two year old man, was admitted on June 3, 1955,

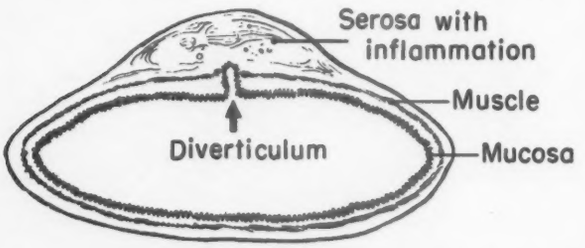

FIG. 2. Cross section of cecum and mass drawn to actual size. The mass which was felt in the anterior cecal wall was actually the result of an inflammatory reaction, especially involving the serosal component, resulting from a tiny diverticulum projecting a short distance into the wall from the mucosal side.

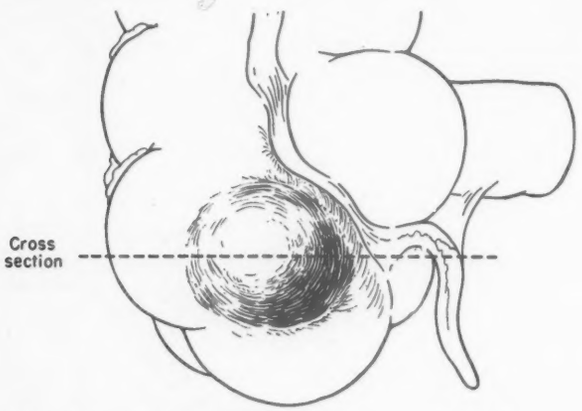

FIG. 3. Appearance of cecal mass at surgery. Mass measured 3 cm. in diameter and 2 cm. in thickness. The serosa was intact; neither ostium nor fecalith was palpable.

with pain in the right lower quadrant and nausea of twenty-four hours' duration. He had not experienced vomiting or diarrhea, and had never had a previous episode. His temperature was 99.2°F. There was 3 plus tenderness in the right lower quadrant. White blood cell count was 17,000 per cu. mm., with 95 per cent polymorphonuclears. The preoperative diagnosis was acute appendicitis.

At surgery the appendix was normal. A 2 cm. hard tumor was felt in the lateral wall of the cecum. No projection within the lumen could be palpated and the serosa of the cecum overlying the mass was intact. Early carcinoma of the cecum was diagnosed. Neomycin, 2 gm., was placed in the terminal ileum. Right colectomy was performed.

Pathologic examination revealed a 2 cm. long diverticulum within the wall of the cecum. The

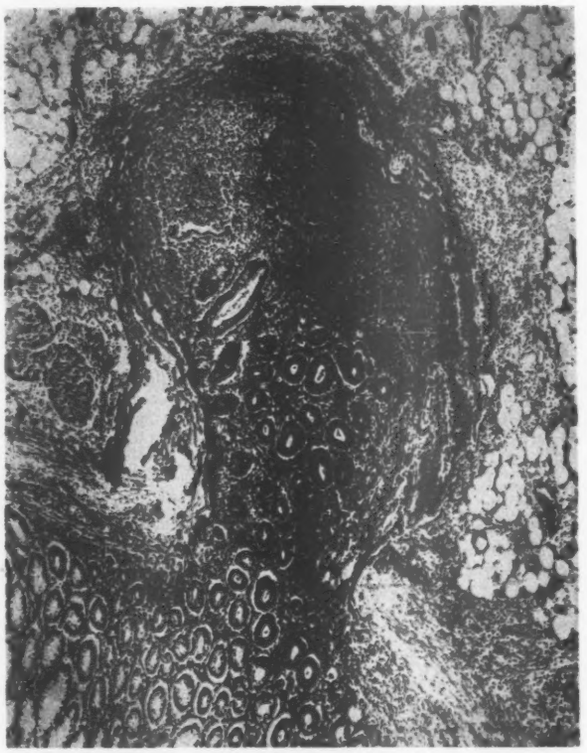

FIG. 4. Case II. Cross section of cecal wall containing an inflamed diverticulum. The mucosa is shown at the bottom of the picture extending upward into the diverticulum. The apex of the diverticulum is within the serosal layer. The muscular layer running through the center of the photograph does not extend around the diverticulum.

ostium and lumen measured 5 mm. in diameter and was surrounded by inflammatory reaction within the wall of the cecum. Mucosa was present; no muscle layers were visible. The patient was well one month postoperatively.

CASE III. A. P. (Hosp. No. 1243-170), a thirty-three year old woman, was admitted on November 7, 1950, with upper abdominal discomfort, more to the right, of forty-eight hours' duration. She did not experience vomiting, but had some anorexia. Bowel movements were normal. Her temperature was 100°F. There was tenderness in the right lower quadrant; no mass was noted. Hemoglobin was 8.6 gm.; a transfusion was given. The preoperative diagnosis was acute appendicitis.

At surgery the appendix appeared normal. A mass was felt in the ascending colon. The McBurney incision was closed and a right rectus incision made. A rubbery, 6 by 8 cm. mass was felt in the wall just above the cecum. It was surrounded by edema but had an intact serosa. No diverticulum could be seen. The diagnosis was carcinoma. Right hemicolectomy was performed.

Pathologic examination showed a 3.2 cm. mass

in the mesenteric side above the cecum. In the center, leading from the lumen, was a small mucosal-lined cavity with surrounding thickened colon wall. Microscopic section showed mucosa. No muscle layers were noted. The patient was discharged in two weeks. She was re-admitted three times in the next six months for obstruction and abscess. When last seen on June 23, 1955, she was entirely well.

CASE IV. D. B. (Hosp. No. 1094-992), a thirty-nine year old man, was admitted on July 22, 1948, with abdominal cramps of two days' duration. No other illness was noted. He had been constipated and was taking laxatives. His temperature was 100°F. White blood cell count was 12,000 per cu. mm. The abdomen was moderately distended with tenderness in the right lower quadrant. The preoperative diagnosis was acute appendicitis.

At surgery on July 22, 1948, the appendix was normal. There was a mass with some superficial inflammatory reaction involving the cecum and ascending colon. It did not protrude within the lumen and could not be distinguished definitely from malignancy. No diverticulum was seen on the surface. Ileotransverse colostomy was performed. On August 30, 1948, right colectomy was performed, although at that time no lesion could be palpated.

Pathologic examination showed that the mucosa of the cecum formed shallow diverticula-like pouches which did not extend through the muscularis. Otherwise, the appendix and colon revealed no abnormalities. The patient was well two years later.

CASE V. P. Q. (Hosp. No. 1218-888), a thirty year old man, was admitted on June 25, 1950, with abdominal pain and vomiting of three days' duration with pain in the right lower quadrant. There was no history of a previous attack. Appendectomy had been performed eleven years previously. His temperature was 100°F. White blood cell count was 8,400 per cu. mm. There was tenderness in the right lower quadrant, and the abdomen was distended. The preoperative impression was acute obstruction of the small bowel.

At surgery there were three 5 mm. areas of gangrene with tiny perforation on the anterior surface of the cecum. Carcinoma was considered as a possible cause. No diverticulum could be seen. The cecal wall was somewhat thickened. Streptomycin, 2 gm., was inserted in the colon. Right colectomy was performed.

Pathologic examination revealed a pocket in the anterior cecal wall measuring 1.5 cm. in diameter and 2 cm. in depth. The cecal wall was 1 cm. thick and inflamed. Microscopic examination showed diverticulum with necrotic mucosa; no muscle. The patient was discharged in ten days. Six-month follow-up revealed no gastrointestinal symptoms.

CASE VI. L. E. (Hosp. No. 1522-643), a thirty-four year old woman, was admitted on October 5, 1954, with an eleven-year history of amebic colitis,

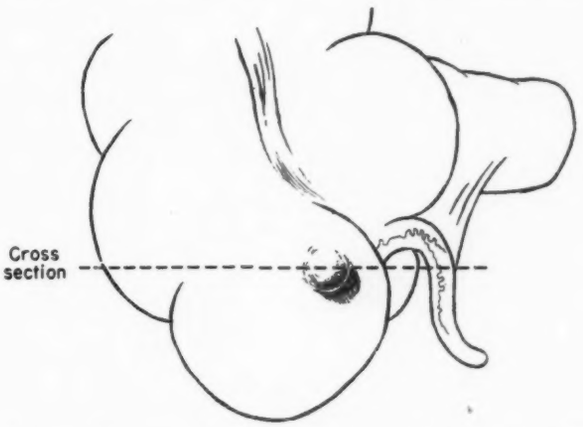

FIG. 5. Case VII. Appearance of usual type of cecal diverticulum. The finger-like projection extending above the surface of the cecal wall is obvious. There was minimal inflammatory involvement of the cecum. Cross section of the diverticulum is indicated by the dotted line, showing serosa and mucosa but no muscle fibers.

never proved, but treated elsewhere. The patient was anemic and weak, and had lost 70 pounds in the last four years. She complained of diarrhea. Barium enema showed almost complete obstruction in the ascending and transverse colon. Operation was performed on October 23rd for probable carcinoma with obstruction, and a thickened, hard area in the ascending colon was felt. Hemicolectomy was performed.

Pathologic examination revealed a small diverticulum in the ascending colon with much fibrosis, scarring and stenosis. In addition there were several diverticula in the cecum, 1 cm. in depth and ulcerated. The microscopic diagnosis was diverticulum with a question of amebic ulcers, although no ameba could be demonstrated anywhere in the specimen. Follow-up one year later revealed that the patient was well.

THE USUAL DIVERTICULUM

In the cecum and ascending colon the usual diverticulum in our series (eight new cases, Table v) protrudes above the serosal surface and can be recognized readily as an inflamed tiny finger tip–like projection above the bowel wall. (Fig. 5.) This type of diverticulum is usually evident to the surgeon even though he has never seen one before and is not familiar

TABLE V

ACUTE CECAL DIVERTICULITIS OBVIOUS AT EMERGENCY SURGERY

Case	Description	Site	Procedure Performed	Course	Result
VII	1 by 1 cm. discolored diverticulum	Anterior	Excision and closure	Uneventful	Well 1 yr.
VIII	1.7 by 1 cm. inflamed diverticulum	Lateral	Excision and closure	Uneventful	Well; dead 2 yr. later of brain hemorrhage
IX	1 cm. inflamed diverticulum	Posterior	Excision and cecotomy	Uneventful	Well 3 yr.
X	Perforated tiny diverticulum	Anterior	Excision and closure	Uneventful	Well 1 yr.
XI	1 by 1 cm. inflamed diverticulum	Anterior	Inversion and closure	Uneventful	Well (mild distress) 5 yr.
XII	1 by 3 cm. necrotic diverticulum	Lateral	Excision and closure	Uneventful	Well 1 yr.
XIII	Multiple tiny inflamed	Lateral	Opened and cecostomy	Uneventful	Well 1 mo.
XIV	Inflammatory thickening, later proved diverticulum by enema	Medial	None	Uneventful	Well 5 yr.

with the entity. This was true of the eight cases in this report. (Table v.) The sixty-four cases of this variety (Table II) recorded in the recent literature, in the main, were identified easily. All of our cases of this type and the majority of these in the literature (95 per cent) were treated by a simple procedure such as excision and closure of the cecal wall.

USUAL TYPE OF CECAL DIVERTICULUM

Appendectomy was performed in each case if not done previously, and all appendices were normal pathologically.

CASE VII. A. P. (Hosp. No. 1410-550), a thirty year old woman, was operated upon in March, 1954, for acute appendicitis. A 1 by 1 cm. reddish black, projecting diverticulum was excised from the anterior aspect of the cecum and the wall was closed. The patient was well in February, 1955.

CASE VIII. A. L. (Hosp. No. 850-598), a sixty-one year old woman, was operated upon for acute appendicitis in February, 1954. An obviously acutely inflamed diverticulum on the lateral cecal wall was excised and the opening closed. Tube cecostomy was also performed. Pathologic report revealed a 1.7 cm. diverticulum, no muscle fibers. The patient had no further gastrointestinal symptoms. She died of a subarachnoid hemorrhage in December, 1955.

CASE IX. N. C. (Hosp. No. 1426-830), a sixty year old woman, was operated upon for acute intestinal obstruction in August, 1953. A 1 cm. inflamed diverticulum was excised with the appendix at the ileocecal junction and a cecostomy tube inserted. A 3 by 7 mm. inflamed diverticulum without muscle layers was described by the pathologist. The patient was well three years later.

CASE X. E. H. (Hosp. No. 1542-978), a thirty-eight year old man who had had a previous appendectomy, was operated upon with a mistaken diagnosis of a ruptured ulcer in December, 1954. There was general peritonitis and a mass in the cecum, on the surface of which projected a tiny inflamed diverticulum. This was excised and the wall closed. Pathologic examination showed inflammation of a tiny portion of the "cecal wall." This must represent the diverticulum with muscle in the wall. The patient was well one year later.

CASE XI. C. R. (Hosp. No. 807-321), a thirty year old woman, was operated upon for acute appendicitis in June, 1951. A 1 by 1 cm. inflamed diverticulum was inverted at the base of the cecum. She was followed up for five years and had occasional mild distress in the right lower quadrant of the abdomen.

CASE XII. B. L. (Hosp. No. 1580-405), a sixteen year old boy, was operated upon for acute appendicitis in May, 1955. A 3 cm. mass was freed up on the lateral cecal wall and recognized easily as a necrotic diverticulum. It was excised. Microscopic examination showed mucosa but no muscle layer. The patient was well fourteen months later.

CASE XIII. R. W. (Hosp. No. 1606-520), a forty-nine year old man, was operated upon for acute appendicitis in September, 1955. A necrotic diverticulum on the anterior cecal wall was excised and a de Pezzer catheter inserted. The patient did well and was discharged on the sixth day. No pathologic specimen was obtained. He was well one month later.

CASE XIV. A. D. (Hosp. No. 1262-286), a forty-five year old woman, was operated upon for acute appendicitis in February, 1951. An inflammatory area 3 cm. wide was seen and felt on the medial

cecal wall. A normal-appearing appendix was removed. Barium enema x-ray four months later revealed a 3 cm. diverticulum of the cecum. The patient was well five years later.

COMMENTS

It is common for the surgeon and pathologist to stress the congenital [*6,25,30,58*], solitary, finger tip–like nature of cecal diverticula in contrast to the multiple, tiny, fat-covered, acquired diverticula frequently observed in the sigmoid colon. Many striking reports and pictures of the solitary protruding diverticulum of the cecum and its comparison with a Meckel's diverticulum have left in the mind of many a preconceived notion that this is what to expect. It will be helpful, therefore, to realize that 44 per cent of the cecal diverticula in this report and 33 per cent of those recorded in the recent literature bear no resemblance to this preconceived notion. These cases of diverticulitis produced a mass within the wall of the bowel which by inspection, palpation and superficial dissection could not be differentiated from carcinoma until the specimen was removed for pathologic examination. This group of cases needs a distinguishing name to differentiate them from the usual variety of diverticulitis. They present an entirely different picture for the surgeon and pathologist, which in the past has not been sufficiently emphasized. No surgeon can become personally familiar with many cases of this type and must understand the entity through the recordings of others. The authors propose that this variety be termed "hidden" diverticula to distinguish them from the more usual type.

In these cases no simple surgical procedure was possible which would deal with the problem adequately. Direct biopsy [*49,60*] into the mass through the bowel wall and into the lumen is unsatisfactory and leaves the mass, if inflammatory, untreated. There is, in addition, an element of uncertainty in the mind of the surgeon as to whether or not he has obtained an adequate biopsy specimen to rule out carcinoma. Opening the bowel at a distance away from the mass and inspecting the undersurface carries with it the same objections. Confronted with such a situation, it seemed reasonable to the authors to perform immediate resection of the right colon and now, after review of the problem, no better solution has been found. Therefore, if the patient's condition is satisfactory and the surgeon is prepared, an immediate resection is advocated. Under these conditions, the use of 5 gm. of neomycin dissolved in 500 cc. of saline and injected intraluminally prior to resection, as suggested by Poth [*60*], is worth while. In six cases treated by resection in this series there was no mortality, and in the thirty reported in the literature the mortality was 3 per cent.

Carcinoma of the cecum and right colon encountered at emergency surgery is a closely allied condition. In 15 per cent of the 885 cases reported by Mayo [*32,33*], prior appendectomy had been performed after the onset of symptoms referable to the malignant process, probably indicating the acute symptoms caused by the cecal carcinoma had required emergency surgery but the carcinoma had not been discovered. It is likely that a mass was present at the initial surgery in these cases but was not, or could not, be palpated. A series of seventeen cases of carcinoma producing emergency abdominal symptoms has been reported recently by Patterson [*37*]. This study and those of others [*11,15,22,38,51*] suggest strongly that if early carcinoma of this region is to be discovered, the surgeon performing emergency surgery must include it in the differential diagnosis at the operating table. When the appendix does not appear to be a satisfactory explanation for the acute symptoms, a Meckel's diverticulum, pelvic disease, ureteral stone, mesenteric adenitis, etc. are looked for, but the cecum may not be carefully palpated. This should be a part of our practice and teaching. However, it appears to be just as important to look for carcinoma of the cecum as a cause of true acute appendicitis since twelve of seventeen recent cases reported by Patterson had, in addition to the carcinoma, acute or gangrenous appendicitis. Amoebic granuloma [*20,31,55*], tuberculoma, terminal ileitis, periappendiceal granuloma [*59*], endometriosis, etc. may occasionally be a part of the differential diagnosis at surgery.

In discussing diverticlitis of the cecum it is important not to confuse the hidden type with the more usual variety of this disease in which the diverticulum-like nature of the process can be recognized by the surgeon at the operating room table. There can be no differences in opinion as to the proper treatment of the "usual" easily recognized variety. In each of the eight cases in this report and in almost all of the sixty-four cases recorded in the recent

literature, a simple surgical procedure was performed. This usually consisted of simple excision and closure and is usually all that is needed. The mortality in the present series was zero; in the literature it was 5 per cent with this form of the disease.

SUMMARY

About one-third of all cases of acute cecal diverticulitis will be hidden within the wall forming a mass indistinguishable from carcinoma until the resected specimen is examined carefully. A tiny diverticulum measuring no more than 1 mm. in diameter may be found. Statistically this type of lesion has occurred once in 1,100 emergency celiotomies for acute conditions of the abdomen. If the patient's condition is satisfactory and the surgeon is prepared, resection of the right colon is advised. For the usual obvious variety of cecal diverticulitis, simple excision and closure is all that is necessary.

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DISCUSSION

Joel Baker (Seattle, Wash.): I congratulate Drs. Greaney and Snyder for their excellent presentation of a condition that is rare in the individual surgeon's experience, yet constitutes a rough hurdle in judgment when encountered at emergency operation.

At the Mason Clinic we have had five cases of acute solitary cecal diverticulitis, two having been previously reported. All were in males, all presented as emergencies, and all were thought to be appendicitis.

At surgery three were recognized as solitary diverticulitis; simple diverticulectomy was performed at the time in two and deferred in the third until the inflammation subsided, a staging more recently recommended by Welch, but for which I believe the need will not too often arise. In the remaining two cases the entire cecum was resected, in one because of the extent of the cecal involvement, and in the other because it could not be differentiated from carcinoma. In this instance a radical cancer operation was performed. This case would fall into the classification of the "hidden" type of diverticulum discussed by the authors, in which the character of the mass can be distinguished only at pathologic examination. There were no deaths in this small series and none in their fourteen cases.

It seems to me that a preoperative diagnosis of acute cecal diverticulitis will continue to be difficult to obtain. When these acute emergencies present, even if we think of this possibility, one is loathe to hazard an x-ray of the colon which requires careful bowel preparation for satisfactory interpretation. If we could, with assurance, make the diagnosis preoperatively, as in the case of a previously demonstrated cecal diverticulum, there would be an advantage in giving the inflammatory process a chance to subside and in preliminary bowel preparation. I am afraid this advantage will rarely prevail.

The more practical difficulty is in establishing the diagnosis at laparotomy, without the aid of histologic examination, for technical reasons, and applying an adequate minimum of corrective surgery. Lauridsen and Ross, in reviewing 153 acute cases, found a mortality of 1.6 per cent after simple diverticulectomy as against 7.3 per cent when the cecum was resected. I have forgotten how this compares with the rate reported to this Society several years ago by Dr. Boyden for primary one-stage resection in acute sigmoid diverticulitis. With present day antibiotics and the precautions outlined by the authors, this figure should be lower today.

When cancer cannot be excluded, the entire right colon and mesenteric lymphatics must be resected, and staging will not often be desirable.

In the one operation we staged, the patient was young and the diagnosis certain. When the diverticulum was excised, it proved histologically to

have all three layers in its wall, thereby supporting the statement of the authors and others that a diverticulum in the cecum may differ from those in the left colon in that they may be true congenital diverticula rather than simple herniations of the mucosal layer.

Last year Dean Seabrook presented us with a rare cecal lesion to be considered in the baffling bleeder—the fatty infiltration of the ileocecal valve. This year Drs. Greaney and Snyder have given us another cecal lesion to consider in emergency conditions of the abdomen. Incidentally, these patients may also bleed. As a corollary, when in our routine examinations solitary or scattered tics are found in the right colon, the patient should not only be alerted as to their presence but also prompted to relay voluntarily this information to any physician who might see him for an acute abdominal crisis. This possibility could not then be overlooked, and these few would realize a dividend from their prior routine examinations.

Nelson J. Howard (San Francisco, Calif.): I found the paper of Drs. Greaney and Snyder most interesting. We are quite used to thinking of acute diverticulitis of the sigmoid in patients with left-sided abdominal pain, fever, tenderness and a palpable tender colon. We tend to forget that diverticulitis of the cecum or ascending colon may occur. If one tabulates the ages of the patients reported in the authors' paper and those from the collected literature, it is found that the majority are under fifty years of age. It is easy to see why in this age group the disease is usually taken for appendicitis. However, from the authors' studies it is apparent that we must keep in mind acute diverticulitis when patients present with symptoms in the right lower quadrant of the abdomen. The lesion which they term hidden diverticulitis occurs, I am sure, in the sigmoid as well, but because of the number of diverticula usually involved it is more easily recognized. I have operated upon two patients with the hidden type of diverticulitis of the cecum and have found two more cases of the disease in the files of St. Joseph's Hospital in which operation was performed; one was the projecting, finger-like type. All four of these patients were younger than fifty; two were in their twenties. Except when the diverticulum is obviously finger-like and projecting, the lesion bears a resemblance to carcinoma, as do the amebic granuloma and the hyperplastic type of tuberculosis. The swelling, induration and glandular enlargement make it imperative to resect the lesion so as to be sure that carcinoma is not present, and as Drs. Snyder and Greaney state, no better treatment than resection can be offered.

R. Bruce Henley (Oakland, Calif.): I, too, would like to congratulate Drs. Greaney and Snyder on this very fine paper.

I have had eight of these cases in my own practice during the past seven to eight years. A short history of symptoms varying between twenty-four to seventy-two hours occurred in all patients, and the preoperative diagnosis was uniformly acute appendicitis. In two of the patients the abdomen had already been opened when I was called.

I would classify the first five cases as examples of the hidden type of diverticulitis of the cecum because the area of inflammation in the mass was much larger than a simple diverticulum. In three of the five cases, the inflammatory mass measured from 1 to 3 inches in diameter and we elected not to perform any definitive surgery. Because we could not rule out carcinoma in the two other cases one-stage right colectomy was performed.

There were two patients with acute diverticulitis of the hepatic flexures. The inflammatory masses were freely movable but the size was such that resection of the colon would have been required for removal, and not simple diverticulectomy. No definitive surgery was performed in these two cases.

Acute diverticulitis in a low-hanging transverse colon with a large inflammatory mass was present in the last patient. The operator had made a preoperative diagnosis of acute appendicitis. When I arrived in the operating room I could not rule out carcinoma completely, so resection of the colon was performed.

In three of the eight cases resection of the colon was performed with uneventful recoveries. No definitive surgery was deemed necessary in five cases. All five made uneventful recoveries and follow-up barium enemas were negative.

W. Kenneth Jennings (Santa Barbara, Calif.): I would like to bring to your attention still another unusual surgical emergency involving the cecum, which in this case was indistinguishable from acute diverticulitis.

This last summer we removed the cecum from a sixty-nine year old woman who was admitted to the hospital complaining of acute right-sided pain of about nine hours' duration. There was nothing of significance in her past history except that at age sixty-two she had undergone appendectomy for a perforated appendix.

At the operating table we were unable to distinguish between acute diverticulitis and carcinoma with secondary infection involving the cecal wall, so right hemicolectomy was performed.

Upon opening the bowel we found a lesion which involved about one-third of the cecal mucosa and extended into the terminal ileum.

There was ulceration and extensive secondary infection affecting almost the entire wall of the cecum and the terminal ileum.

Microsections of the lesion revealed a villous adenoma. As you know, villous adenoma is common in the rectum and the lower sigmoid, as are diver-

ticula, but is rarely seen in the right colon. This was the first one in our experience, and we thought it might be worth presenting.

LOUIS P. GAMBEE (Portland, Ore.): A number of years ago a physician in a small community subjected a man to laparotomy because he believed the man had acute appendicitis. He found, however, that the appendix was not involved and that there was a small tumor mass proximal to the appendix. He biopsied this mass and was told that it was an inflammatory process.

This patient sought no further attention for seven years when he finally was sent to me with a palpable tumor mass in the right lower quadrant of the abdomen. This tumor mass proved to be a carcinoma which originated in a diverticulum of the cecum.

CLARENCE E. STAFFORD (Los Angeles, Calif.): I would like to add one further comment to this very informative paper.

A forty-seven year old Japanese man had a massive gastrointestinal hemorrhage as a result of inflammatory change in the base of a diverticulum of the right colon just above the cecum.

From the specimen one could recognize the appendix on the right and the ascending portion of the colon on the left. There were three diverticula; in the center one the inflammatory changes at the base had involved a major blood vessel, which was the source of bleeding. Because of the involvement of the blood supply, it was necessary to resect a portion of the right colon.

This case adds another complication that one has to think about when looking for the cause of gastrointestinal bleeding.

GORDON K. SMITH (Los Angeles, Calif.): I would like to make one point in line with the paper by Dr. Goodwin on "The Surgeon, the Urologist and the Patient with Abdominal Pain."

The first case of diverticulitis of the ascending colon that I encountered was during my residency.

A young boy, about seventeen years of age, was sent to my Chief because his condition was going downhill after three unsuccessful attempts to drain a perinephric abscess on the right side.

When he arrived at the hospital it was obvious that the drainage was inadequate. We enlarged it and found some packing present. About the second day, as the packing was removed, fecal matter came out with it.

The diagnosis then became quite obvious, and a barium enema revealed a perforation of the ascending colon just above the tip of the cecum. At laparotomy, right colectomy was performed.

I think this is an important point to remember because diverticula in the cecum and ascending colon can perforate posteriorly through the leaves of mesentery.

EDWARD M. GREANEY (closing): We gave a great deal of attention to the problem of whether or not these diverticula were true or false. After reviewing all available slides with the pathologist, the conclusion was reached that even though muscle was demonstrated in the section, these were still acquired or false diverticula. This remains, however, a controversial issue.

Diverticulitis of the Colon

RADICAL VS. CONSERVATIVE TREATMENT

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As the average age of the population increases, it is to be expected that more diverticulosis of the colon will be seen and more of the complications of the condition will be encountered. In the past, surgery was performed mainly for the complications but in recent years surgeons have adopted a more vigorous approach to the treatment of diverticulosis and diverticulitis [2,4,13]. With the aid of antibiotics and improved surgical and anesthetic technics, the mortality rate for resections of the colon has become quite low and it is possible to remove relatively large portions of the colon with little risk to the patient [3,6,14]. Many doctors are apparently not aware of this fact and have continued to treat the condition by "conservative" medical means because they believe more "radical" treatment is not justified.

It would be possible to reach some agreement as to the best treatment if the prognosis or response to treatment could be determined in any given case at the outset. Unfortunately, this is not possible. This study was undertaken in an effort to find evidence which might help in determining the best type of treatment to be employed.

TABLE I
AGE DISTRIBUTION

	No. of Cases	Average Age
Diverticulosis:		
Male	142 (40.3%)	67 yr. (34 to 90)
Female	211 (59.7%)	67½ yr. (23 to 92)
Total	353	
Diverticulitis:		
Male	64 (35.7%)	65 yr. (41 to 83)
Female	115 (64.2%)	66 yr. (39 to 91)
Total	179	

TABLE II
DISTRIBUTION OF DIVERTICULOSIS

Distribution	Per cent
Sigmoid colon only	48 } 72
Descending and sigmoid colon	24 }
Transverse descending and sigmoid colon	10
Entire colon	17
Cecum only	1

A group of 353 cases of diverticulosis of the colon seen in the San Francisco Hospital between July, 1946 and July, 1956 was reviewed. All patients seen on both the Stanford and the University of California services were included. Many had had previous entries for the same condition but returned during this period for treatment. A diagnosis of diverticulitis was made in 179 (50.7 per cent) cases. (Table I.)

The diagnosis of diverticulosis was made by roentgenologic examination and by autopsy. Many cases were found coincident with some other condition. The youngest patient in whom diverticulosis was found was a Chinese girl of twenty-three years who died of tuberculosis. Many of the patients were more or less obese although diverticulosis was found in some who were very thin. About 15 per cent were listed as being markedly obese. The condition was distributed throughout the colon as shown in Table II.

The diagnosis of diverticulitis was made clinically in most cases with confirmatory roentgenographic evidence, while in other cases the diagnosis was made at surgery or at autopsy. The main symptoms were dull abdominal pain, "fullness" in the abdomen, gas, rectal urgency and diarrhea in many cases. Several gave a story of generalized abdominal pains becoming localized in the left lower quadrant of the abdomen after eight to twelve hours. Vomiting occurred in many of the severe cases. The average temperature of those who were diagnosed clinically as having diverticulitis was 100.6°F. and the average white blood count was 14,350 per cu. mm. In several of the more elderly patients there was little constitutional reaction, and in four such patients with a ruptured diverticulum and peritonitis there was only low grade fever and a normal leukocyte count.

Obesity makes localization of the tenderness difficult and may mask some of the physical signs. Since most diverticulosis and diverticulitis is found in the sigmoid colon, most of the tenderness is found in the left lower quadrant of the abdomen. However, the sigmoid colon often lies deep in the pelvis or may extend to the right side, and thus may alter the localization of tenderness. This may account for the fact that a diagnosis of diverticulitis is often missed or is difficult to establish. Three of the patients in this group had acute diverticulitis in the cecum and another had diverticulitis in the transverse colon. In each of these cases, the maximum tenderness was at the site of the involved area.

There were fifteen acute perforations with peritonitis in this series although it has often been stated that they are rare. One was large enough to admit the index finger without difficulty. One interesting case involved a patient who had a perforation which occurred while he was receiving cortisone therapy for scleroderma; the symptoms and signs were masked. The cause of death was found at autopsy. There were no perforations of diverticula due to foreign bodies in this series. An interesting perforation seen recently in private practice was due to a chicken bone. At surgery one end of the bone was caught in a diverticulum and had perforated it.

In those patients with a discharge diagnosis of acute diverticulitis during this ten-year period, it was found that 65 per cent were admitted on the Medical Service while 32 per cent were admitted on the Surgical Service and 3 per cent on the Gynecological Service. This fact led to an investigation of the admission diagnoses which indicated that diverticulitis can simulate a variety of conditions. (Table III.)

TABLE III
ADMISSION DIAGNOSES

Diagnosis	No. of Cases
Diverticulitis	52
Rectal bleeding	26
Neoplasm of colon	22
Bowel obstruction	21
Enterocolitis	13
Abdominal pain	12
Appendicitis	7
Peritonitis	6
Urinary tract infection	5
Twisted ovarian cyst	4
Pelvic abscess	4
Dissecting aneurysm	2
Pancreatitis	2
Ulcer	2
Cholecystitis	1
Total	179

Most of those patients admitted on the Medical Service and in whom complications developed were seen later by a surgeon in consultation, but the largest percentage were treated with a conservative medical regimen. In most instances, after a diagnosis of diverticulitis was made, the patient was given a low residue diet and antispasmodic medication. Antibiotics or sulfa drugs were usually given if fever or leukocytosis was present. If the process subsided without complications, the patient was discharged on a low residue diet and was told to return to the outpatient clinic if he had recurrent symptoms. Some of these patients apparently had no further trouble, but many had repeated entries for the same condition and in some severe complications subsequently developed. Occasionally patients with inflammatory masses were treated conservatively without surgical consultation and no active treatment was advised for the purpose of eradicating the condition. This is not pointed out to criticize the treatment of any particular patient in this series but rather to indicate the unfortunate attitude of many who see patients with this condition.

Diverticulosis is a condition which is found

mainly in the older age groups with the number and size of the diverticula frequently increasing as the person becomes older. The incidence of diverticulosis is generally considered to be about 10 per cent in those above the age of forty years. It has been estimated that diverticulitis will develop in about 10 to 20 per cent of those patients with diverticulosis; and of those with diverticulitis, about 10 to 20 per cent will require surgical intervention.

TABLE IV
DIVERTICULITIS

Follow-up (yr.)	No. of Cases
1 to 5	61
5 to 10	30
10 to 15	11
15 to 20	7
Total	109

An attempt was made to determine the course of the patients seen in this series. There was not a sufficient number of cases of asymptomatic diverticulosis with adequate follow-up to justify any conclusions. However, it was interesting to find that of twenty-three cases in which barium enemas originally showed only diverticulosis, during the ensuing five to twenty years, diverticulitis developed in ten; three had episodes of bleeding for which no cause other than diverticulosis was found; and in two, perforation with abscesses developed.

Of the cases of diverticulitis, only those were included in which it was considered there was adequate objective and subjective information which might allow some conclusions to be made. Table IV shows the number of cases followed up.

The number of patients with diverticulitis having progressive changes, as opposed to those who had no further attacks or changes as determined by recheck, x-ray examination or follow-up histories, is shown in Table V. It should be emphasized that these statistics apply to patients who originally had complaints and signs referable to their bowel severe enough to warrant hospitalization and in whom a definite diagnosis of diverticulitis was made.

These cases were divided into two main groups with the one- to five-year group indicating the early changes and the five- to twenty-year group indicating the late changes. It can be seen that as time progressed a higher percentage of patients who had once had diverticulitis either had repeated attacks or complications developed. Of the 109 patients making up both groups, seventy-four (67.9 per cent) had recurrent symptoms which required additional hospital entries. Of these, thirty (40.5 per cent) patients had recurrent attacks of diverticulitis while complications developed in forty-four (59.4 per cent). Certainly, some of the patients with progressive changes might have been saved additional periods of disability and some of the serious complications if the involved segment of bowel had been removed as an elective procedure earlier in the course of the disease.

TABLE V
FOLLOW-UP OF PATIENTS WITH DIVERTICULITIS

Progressive Changes	One to Five Years (No. of Cases)	Five to Twenty Years (No. of Cases)	Total Cases
Recurrent diverticulitis	12	18	30
Perforation, abscess, fistula	10	12	22
Stricture or obstruction	4	7	11
Hemorrhage	9	2	11
Total	35 (57.4%)	39 (81.3%)	74 (67.9%)
No changes (one attack)	26 (42.6%)	9 (18.7%)	35 (32.1%)
Total	61	48	109

The progress of this condition is shown in several case summaries which follow.

CASE REPORTS

CASE 1. M. M., a seventy-three year old woman, was seen in May, 1945, complaining of recurrent attacks of lower abdominal pain of ten years' duration. A barium enema showed diverticulosis of the sigmoid colon. She was put on a medical regimen but returned in September, 1946, again complaining of lower abdominal pain. The barium enema was about the same as in 1945 and she was again put on a medical regimen. In July, 1947, she had a hemorrhage from the bowel. Barium enema showed signs of diverticulitis of the sigmoid colon with narrowing of the bowel lumen. She was given a finely divided diet and antispasmodics. She was seen in October, 1949, complaining of constant abdominal pain. Barium enema showed signs of diverticulitis with obstruction in the sigmoid colon. Resection of the sigmoid colon was performed.

CASE II. E. A., a sixty-one year old woman, was first treated in 1943 in the outpatient clinic for recurrent attacks of pain in the left lower quadrant of the abdomen. In 1945 a barium enema was interpreted as showing diverticulitis of the sigmoid colon. In December, 1948, an inflammatory mass was noted in the left lower quadrant of the abdomen. Transverse colostomy was performed in January, 1949, but a pelvic abscess developed which was drained through the rectum in March, 1949. Her condition improved but in July, 1949, she returned with a sigmoidovaginal fistula. Resection of the sigmoid colon was performed.

CASE III. A. H. S., a thirty-six year old man, was seen in 1943 with diverticulitis of the sigmoid colon with an abscess. This was drained and he left the hospital after three months. He was seen again in December, 1948, with a tender mass in the left lower quadrant of the abdomen which subsided with bedrest and penicillin. He returned in March, 1949, with another mass in the left lower quadrant and a barium enema showed signs of diverticulitis of the sigmoid colon with obstruction. The mass subsided again with the aid of penicillin but he returned in June, 1949, with lower abdominal pains. A barium enema at that time showed a sinus tract outside the sigmoid colon. Resection of the sigmoid colon was performed.

CASE IV. M. A. W., a fifty-seven year old woman, was seen in 1939 after passing a large amount of blood per rectum. A barium enema showed diverticulosis of the sigmoid colon. She returned in October, 1940, complaining of left-sided abdominal pain, and a barium enema again showed diverticulosis of the sigmoid. In August, 1944, she had a bowel obstruction. At operation it was found to be due to a loop of ileum which was adherent to a mass in the sigmoid colon due to diverticulitis. She was seen again in February, 1946, complaining of lower abdominal pain. A barium enema showed a constricted area in the sigmoid colon. Left colectomy was performed. She had signs of partial bowel obstruction in August, 1950, and a barium enema showed that diverticulosis of the ascending and transverse colon had developed.

In addition to these illustrative cases, there were many other patients with numerous entries. One had eight separate entries for diverticulitis over a period of thirteen years, with an average hospital stay of twenty-nine days for each entry. Each time she had typical physical findings of sigmoid diverticulitis with fever and leukocytosis. She was treated by dietary management, sulfa drugs and antibiotics, and although she responded well to the treatment, she spent 232 days in the hospital for this condition and each time ran the risk of having a serious complication. From an economical as well as a risk standpoint, this patient would have done better with resection of the sigmoid colon which was the site of her diverticulitis.

Most of the patients who were treated conservatively over a period of years were seen by different members of the medical staff on each entry. This was due to the rotation of services and may have contributed to the fact that a large number had repeated entries without consideration of more radical treatment.

ASSOCIATION OF DIVERTICULITIS AND CARCINOMA

Frequently it is difficult to differentiate between the signs and symptoms of diverticulitis and carcinoma of the colon. A number of criteria have been proposed in helping to make a differential diagnosis between these two conditions [*12*]. Usually, when there is any question, the patient is put on a low residue diet with antispasmodics for a period of three or four weeks and then the barium enema is repeated. Frequently, a definite diagnosis still cannot be made at that time so surgery is advised. There will probably always be some question as to the correct diagnosis in any case in which there is constriction of the lumen of the bowel in the presence of diverticulitis and often, even at operation, a definite diagnosis cannot be made without the help of a pathologist. This state of affairs existed in a number of the operated patients in this series, but it was especially interesting to note that unsuspected carcinoma was present in two patients with diverticulitis and was found in the surgical specimen after removal. This also happened recently in a private case in which there was a ruptured diverticulum with a small abscess outside of the bowel, but in the resected portion there was a small carcinoma about 1 inch from the site of rupture which had not been demonstrated by barium enema. There was no indication in these cases that diverticulosis played a part in the origin of the carcinoma. Benign polyps were found in one of the resected specimens in this series, which further illustrates the point that diverticulitis may cause changes in the bowel which can hide other underlying conditions.

Although it is not wise to attempt to resect an acutely inflamed area of bowel, early surgery

is advisable if carcinoma is suspected. The presence of a mass or the signs of obstruction often encourage more rapid action, but too often there is needless delay. If the changes in the bowel due to diverticulitis are severe enough to resemble carcinoma, the patient has a good chance of having persistent trouble and will probably require surgery later anyway, so that little will be gained in most instances by waiting. It is obvious that if a carcinoma is found, a much more radical resection should be performed than if an inflammatory process is found.

BLEEDING

In this series of 353 cases of diverticulosis, sixty-two (17.6 per cent) were admitted because of gross bleeding. This compares with the average of 15 to 20 per cent reported by other observers [5,7,10]. The average age was seventy years (ranging from forty-nine to eighty-eight years); there were twenty-two males (35 per cent) and forty females (65 per cent). In almost all of them there was a practically identical history of a sudden urge to have a bowel movement, often with abdominal cramps, followed by a large bloody stool. Many were awakened during the night with cramps and then passed a bloody stool. Several patients had two or three bloody stools before being admitted to the hospital, and about half complained of weakness and dizziness after passing the bloody stool. In all of these cases no other cause for bleeding was found. Each patient underwent anoscopy and sigmoidoscopy, and all had a gastrointestinal series, which was negative. Most of them also had air contrast enemas. Of course, it is impossible to state with certainty that all patients bled as a result of diverticulosis, but in none was there any evidence that some abnormality in the upper gastrointestinal tract was the cause of the bleeding. In addition, there was no indication of Meckel's diverticulum, polyps, ulcerative colitis, hemorrhoids, blood dyscrasias, etc. Any patient who had roentgenologic changes in the gastrointestinal series or had any constitutional disease which might cause the bleeding was eliminated from this group even though he might have had diverticulosis and a history of bleeding.

Definite signs of diverticulitis were found in twenty-eight patients (45.1 per cent) either by roentgenologic or autopsy findings. Diverticulosis of the sigmoid and descending colon was found in forty-three (69.3 per cent), while diverticulosis of the entire colon was found in nineteen (30.6 per cent). During a seven-year follow-up period, twenty-five patients (40.3 per cent) had recurrent bouts of bleeding requiring from two to four hospital entries. Twenty-one patients required transfusions; twelve required more than 2 units of blood, with a maximum of 5 units for any one patient on one entry. About half of the patients showed signs of bleeding for more than one day, with the average being six days. There seemed to be a definite relationship between the extent of the diverticulosis and the amount of bleeding—those with the most bleeding seeming to have the most advanced diverticulosis.

Two of the patients who were admitted with bleeding required resections two and five years later, respectively, because of diverticulitis with obstructing masses. Three patients had hemorrhages from two to seven days prior to perforation of a diverticulum into the free peritoneal cavity. All three died of peritonitis. In view of these cases, bleeding would seem to indicate an active ulcerative process and the possibility of impending perforation should be kept in mind.

Two patients were operated upon in an attempt to find and control the bleeding. In both, the colon was filled with blood and the remainder of the gastrointestinal tract was normal. Numerous diverticula were present but it was not possible to find the bleeding point by colotomy. The abdomen was closed in each case without doing anything further, and the bleeding subsequently stopped. One patient died of cardiac arrest during resection for an inflammatory mass which had been bleeding.

One patient had had an operation five years before for a carcinoma of the rectum and began to have bleeding from the colostomy. A sigmoidoscope was passed into the colon through the colostomy and a bleeding point on the edge of a diverticulum was seen. The bleeding eventually stopped.

There were two deaths resulting directly from bleeding in this series. An autopsy was not obtained on one, but the other was found to have a large diverticulum about 2 cm. in diameter in the descending colon with "extensive ulceration and inflammation" in its base. Large arteries were seen in the section around the ulcerated area. In another case, bleeding was thought

to be the indirect cause of death in a patient who died with cardiac failure. She was admitted to the hospital with rectal bleeding. She had undergone resection of the sigmoid colon for diverticulitis with a fistula to the vagina one year before, at which time about 18 cm. of bowel had been removed. At autopsy, diverticulosis of the remaining colon was found and in one of the diverticula there was an area of necrosis of the mucosa which was thought to be the site of bleeding.

In reviewing this series of cases with bleeding, it seemed quite evident that there was reluctance on the part of the attending staff to consider the bleeding as due to diverticulitis. This same tendency seems to be almost universal among doctors. It seems that making such a diagnosis is an admission that the doctor is unable to find the cause of bleeding and so a diagnosis of "hemorrhage from the G.I. tract" is made with an additional diagnosis of diverticulosis with or without diverticulitis. Admittedly, it is difficult to find bleeding points in the colon just as it is often difficult to find them in the stomach. However, just because it is difficult to prove does not mean that it does not occur, and more effort should be directed toward definitely establishing this possibility to everyone's satisfaction.

In patients with repeated bouts of bleeding which are apparently due to diverticulosis, resection of the colon should be considered if the condition is localized. This might seem to be a radical viewpoint in patients in the older age group, but hemorrhage is apt to be more severe in patients with arteriosclerotic or hypertensive changes who are frequently found in this group. When massive hemorrhage apparently from the colon persists, exteriorization of the sigmoid colon or a Mikulicz resection should be considered as a possible life-saving procedure.

OPERATIVE PROCEDURES

In the past, diverticulitis has been treated quite conservatively at the San Francisco Hospital. Surgery was performed only for the complications and accounts for the varied procedures and the high mortality rate found in this series. A summary of the operative procedures is shown in Table VI.

Several of the patients operated upon were extremely ill and died in twenty-four hours, but other patients died five to six weeks after surgery as a result of sepsis. A number had complications of renal or cardiac failure. There was only one death among the resections and this was due to cardiac arrest during the procedure. One death occurred several weeks after resection, and autopsy showed death to be due to cardiac failure. The operative mortality in this series is shown in Table VII. These were all complicated cases and account for the high mortality rate.

TABLE VI
OPERATIVE PROCEDURE (FIFTY-SIX CASES OPERATED)

Procedure	Times Performed
Resections	23
Resection with cecostomy	3
Resection with colostomy	8
Exploratory laparotomy (open and close)	8
Closure of perforation	6
Drainage of abscess with colostomy	5
Colostomy only (3 refused closure)	6
Cecostomy	3
Colotomy (for bleeding)	2
Exteriorization of perforation	3
Drainage of abscess only	2
Release of adhesions (obstruction of ileum due to adhesions to sigmoid)	1

TABLE VII
OPERATIVE MORTALITY

Operation	No. of Cases
Resection (cardiac arrest)	1 (4.5%)
Other procedures	12 (35.3%)
Exploratory laparotomy (peritonitis)	2
Closure of perforation (peritonitis)	1
Drainage of abscess and colostomy (sepsis)	3
Colostomy (pulmonary embolus)	1
(sepsis)	1
Cecostomy (peritonitis)	2
Exteriorization (pulmonary embolus)	2

The ideal surgical procedure should be one which will eradicate the condition and, therefore, resection is the procedure of choice. This should be performed usually as an elective procedure. A one-stage resection in an acute case is not advisable. Exteriorization was used for three perforations in this series in which it was believed the affected area could be resected later. Closure of perforations without a proximal colostomy is a hazardous procedure but

good results were obtained in five of six cases. If an acute perforation is reasonably certain in a patient and the general condition is satisfactory, exploratory laparotomy is indicated. The size of the perforation cannot be determined without direct observation. In one case, as noted previously, the perforation admitted the tip of the finger.

Cecostomy was performed as a decompressing procedure in five cases, but colostomy is a better procedure since it will decompress the bowel and also defunctionalize it. When laparotomy is performed and diverticulitis is found, as in seven cases in this series, an evaluation must be made at the time as to the probabilities. If the process seems to be well walled-off and localized only to the bowel, then the area might be reinforced by fastening omentum about it and relying on antibiotics to help the process to subside. If the process is quite extensive and other organs are involved, it is advisable to perform transverse colostomy only and plan resection for a later date

The colostomy may be performed either to the right or the left of the midline, but the transverse colon should be pulled well over to the left so as to leave as much as possible of the colon free on the left side to facilitate the subsequent resection. The time for the resection will vary with each case depending on the extent of the inflammatory process. In addition to the physical signs, the temperature, white blood count and sedimentation rate may help to determine the optimum time for surgery. The second stage can usually be performed after six or eight weeks. If there seems to be a good possibility that carcinoma exists, an attempt should be made to perform the second stage after three or four weeks or as soon as the acute process has subsided.

The mortality rate in most reported series of resections is under 3 per cent. This rate can be maintained if care is taken in selecting patients who are suitable for a one-stage procedure and those who require a multiple stage procedure. The one-stage procedure may be attempted when the bowel can be adequately prepared and when there is no evidence of acute inflammatory process, obstruction, sinus or fistula formation. If at the time of surgery the bowel cannot be adequately mobilized to allow for a satisfactory anastomosis with normal bowel, or if there is increased tension at the suture line, colostomy should be performed at the time of resection. This can usually be closed ten to fourteen days later but should not be done before the patency of the anastomosis has been determined.

In this series, there were fistulas to the abdominal wall in two patients, to the small bowel in two, to the vagina in three, and to the bladder in one. There were sinus tracts outside the bowel in eight patients. In such cases it is advisable to perform transverse colostomy first and resection eight to twelve weeks later.

In most series, as in this one, most of the diverticulitis is found in the sigmoid and descending colon (72 per cent in this series) and when the resection is performed, all of the sigmoid colon and as much of the descending colon as is practical should be removed. There was a recurrence of symptoms in two of the patients in this series in whom small resections were performed. This occurred after two years in one patient and after eighteen years in the other. Both patients showed signs of diverticulitis in the barium enemas, and a small inflammatory mass developed in one.

MORTALITY

There were twenty-seven deaths directly attributable to diverticulitis and its complications. Ten deaths were due to perforations with peritonitis among the non-operated group. The over-all mortality rate was 15.1 per cent.

COMMENTS

In any given case it is impossible to determine the course which diverticulosis will follow. The condition is one which can run a benign course and apparently never cause symptoms, although many times the first symptoms of which the patient is aware may result in severe complications. The first attack of diverticulitis may subside in a few days, may result in abscess formation if a perforation is walled-off, or it may result in diffuse peritonitis and death. According to the experience in this series, if the first attack subsides without complications, about one-third of the patients will have no more trouble, but about two-thirds will have recurrent or progressive changes with over half of the patients having complications. Urinary tract symptoms associated with diverticulitis is a serious sign, especially in the male, since they may be the premonitory symptoms of a fistula between the colon and the bladder.

If surgery is not advised until complications force the issue, it may be necessary as an emergency procedure or it will probably be necessary to perform multiple stage operations [*8,9,11,14*]. If the involved area (sigmoid and descending colon in 72 per cent) is resected as an elective procedure, it can usually be performed in one stage.

Each case should be considered as an individual problem and observed carefully while under treatment. If while under medical management the acute process shows signs of subsiding, one is justified in being conservative. However, after such an attack, the patient should be made aware of the possibility of future attacks and their possible complications.

Resection is considered to be indicated in the following conditions: (1) complications of diverticulitis (obstruction, perforation, abscess, fistula, etc.); (2) recurrent attacks of diverticulitis; (3) persistent deformity shown by barium enema; (4) recurrent massive bleeding from the colon if the diverticulosis is localized; and (5) persistent urinary tract symptoms associated with diverticulitis.

SUMMARY

A study of the cases in this series indicates that diverticulosis is a progressive condition. Once diverticulitis has manifested itself, about two-thirds (68 per cent) of patients so affected will have recurrences; of this group, complications will develop in more than half (59 per cent) over a period of years. Conservative management seems adequate for about one-third of the patients, but it is impossible to tell which cases will have progressive changes and so each case must be followed carefully. Early resection is advocated for patients showing signs of recurrent diverticulitis as well as for its complications. Hemorrhage from the colon may be a sign of impending perforation. Recurrent massive hemorrhages should be considered as a probable indication for resection.

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DISCUSSION

Allen M. Boyden (Portland, Ore.): This paper by Dr. Greene is a most important contribution. During the last decade there has been a growing realization that surgical treatment of diverticulitis has often been inept. The improved results and low mortality rate in a number of recently reported series have led to wider acceptance of certain principles of treatment heretofore considered radical and unsafe. Increasing emphasis is being put on individualization of therapy, and there is less tendency to categorize patients into groups demanding specified methods of treatment. Primary resection after adequate bowel preparation is much more common today. Early resection after colostomy has also markedly reduced the postoperative morbidity of the disease.

Surgeons interested in this disease and its complications agree that indications for resection should be widened to include patients with recurrent diverticulitis, those with associated urinary symptoms and those who show persistent deformity of the involved segment of colon on roentgenographic examination. However, this attitude has been difficult to sell to our colleagues in internal medicine who continue to consider diverticulitis a medical disease until a complication forces their hands. I am unaware of a study which shows so clearly the high incidence of recurrent disease and

eventual complications as this report by Dr. Greene. Several authors have pointed out that approximately two-thirds of the patients treated by surgical measures short of resection, such as drainage of abscess or colostomy with closure, require eventual resection if they are to remain well. Furthermore, the reported late mortality rate of such recurrent and complicated disease has been as high as 12 per cent.

Dr. Greene has shown that in approximately 80 per cent of patients having had one attack of diverticulitis there are severe progressive changes on five- to twenty-year follow-up studies. Were this more widely known and explained to patients, definitive surgery would be more readily accepted. The high mortality rate in the San Francisco hospital series is undoubtedly related to the laissez-faire attitude evident in the case reports listed in this paper.

The high incidence of hemorrhage as a complication of diverticulitis in the series reported by Dr. Greene is of particular interest. Many physicians continue to doubt that serious hemorrhage can result from this cause. Although most series have included a much lower percentage of patients with this complication, it is well recognized that bleeding is not uncommon and may be massive. The problem of treatment is often extremely difficult because a small erosive ulcer may be the source and fail to show on roentgenographic examination or exploration.

Dr. Greene's timely study will prove of real value in helping to crystallize the indications for surgical treatment of colonic diverticulitis. It is unfortunate that it cannot also be published in a journal more widely read by medical specialists in gastroenterology.

Caleb S. Stone, Jr. (Seattle, Wash.): I think this has been an extremely interesting and important paper and I have enjoyed it very much. The remarks by Dr. Boyden were instructive and pertinent.

I wish to limit my comments to the patients characterized by Dr. Greene as those who have recovered from an attack of diverticulitis under conservative treatment without operation. I wish to emphasize the importance of a close follow-up on this group of people.

If these people are closely followed up after recovery, a good many of them can be selected for what we have chosen to call elective resection of the involved segment of colon. Elective resection may be applied in the treatment of diverticulitis but particularly in the prevention of recurrent attacks and the development of complications which follow the original acute attack in a high percentage of cases.

We have selected essentially the same indications for elective resection as Dr. Greene has listed, namely, recurrent attacks; persistent deformity of the colon after clinical recovery; persistent symptoms; occasional or recurrent bleeding; and the occurrence of urinary tract symptoms during or shortly after the apparent recovery from an acute attack of diverticulitis.

If this group of patients who have recovered from an attack of diverticulitis is carefully followed up, a great many of them can be wisely and properly selected for elective resection, thus avoiding the risk of recurrent attacks and the hazards and costliness of complications. This is particularly important since diverticulitis, in general, is a disease seen more frequently as age advances.

The various figures that have been alluded to and the figures on longevity, with which you are all familiar, very clearly indicate that anyone involved in the practice of general surgery today is also heavily involved in geriatric surgery, and our responsibility is not only to cure a disease but also to protect older people from progressive chronic disease which can be cured.

I am sure that if this attitude of close follow-up is practiced, one can attain minimum mortality and morbidity rates in diverticulitis.

Siegfried F. Herrmann (Tacoma, Wash.): I enjoyed this presentation very much, and I am sure that the subject is very important. The presentation was very effective.

My talk before this Society in 1948 was not based on a large series of cases, but I had had some horrible experiences. I knew that diverticulitis could lead to very terrible complications. When a man voids feces or a woman's bowels move through the vagina, that is a very serious situation. However, it can be prevented. That was my message—that an anatomic deformity which becomes involved by inflammatory processes, and then repeatedly becomes involved, is never cured by medical management. In fact, I stated that there was no medical treatment for such diverticulitis. It had to be surgical to be curative.

I also pointed out, on the basis of a few patients, that sometimes I, as well as our radiologists, could not tell the difference between cancer and diverticulitis. Neither could I tell the difference in the operating room when I had hold of the sigmoid, whether this sigmoid harbored cancer or whether it was diverticulitis. Therefore, I said we must operate on these patients so that cancer would not be overlooked.

I pointed out many other errors in diagnosis, and I believed that perhaps I had made a small contribution on the West Coast. However, there was some head-wagging at the time, and I am sure that few of the members present would have chosen me as their surgeon.

This paper was published in the *Western Journal of Surgery, Gynecology and Obstetrics*, and I began

to feel better when my reprints disappeared very rapidly. I had requests from all over the country; but since then I have hardly ever seen my paper mentioned because it was in our Western journal.

I might say also that I have become a little more willing to operate upon patients with diverticulitis because of hidden diverticulitis in the sigmoid.

For example, not too long ago I operated upon the elderly wife of a retired surgeon in Tacoma. This woman had had repeated attacks of abdominal pain which had never been explained, and it was believed that she had some sort of recurring intestinal obstruction.

Although we did not have very good evidence as to where the obstruction was, during another acute episode I was persuaded to operate. I found that she had some thickening in the sigmoid, but that was all. There was no dilated bowel and no real obstruction.

I said, "Doctor, I don't think we'll do any good unless we resect this sigmoid. There's something wrong with it."

The sigmoid contained a hidden diverticulitis, a thickening, without actual projection of a diverticulum.

This woman tells me she lives in a different world. Whereas she had previously been constantly constipated, her bowels now move daily. It is very gratifying to operate upon such a patient, even without the serious complications which we were discussing.

I think there are two different sorts of papers. Sometimes a paper is just based on a small experience and common sense. Then there are papers that are based on more or less astronomic statistics: many, many cases with many percentages. I think those papers are also of value.

Dr. Greene's paper combined both. It is gratifying when the statistics coincide with common sense.

W. Wallace Greene (closing): I wish to thank the discussers.

My only comment is that if this discussion has emphasized at all the dangerous possibilities of conservative management of recurrent cases of diverticulitis, it will have accomplished its purpose

Hyperparathyroidism

A REVIEW OF A COMMUNITY EXPERIENCE IN SEATTLE HOSPITALS

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THE solution of the riddle of the role of the parathyroid bodies in human physiology represents one of the truly great developments of modern medicine. Hidden by their small size and their similarity in texture and color to adjacent thyroid and thymus glands, the parathyroids remained undiscovered throughout centuries of anatomic and surgical dissections until their accurate description by Sandstrom in 1880. Their relatively simple and strikingly independent mission, to control the metabolism of the two minerals phosphorus and calcium, was unsuspected until their extirpation in rats by Erdheim in 1907. Only recently in the laboratories of Dr. Fuller Albright has it become apparent that their primary effect is to control the serum level of phosphorus by controlling its excretion by the kidney, the calcium ion accumulating in the serum in passive response to reduction in phosphorus levels [*21*]. Early clinical recognition of cases followed the brilliant surgical experiment of Mandl (1925) who first transplanted parathyroid tissue into a patient with von Recklinghausen's disease, only shortly to reverse the disease process and the entire theory of the physiology of the parathyroids by excising an adenoma and curing his, the first, patient. Within that year pioneer recognition of the condition followed in this country at the Massachusetts General Hospital and the Mayo Clinic under the stimulus of the physiologic chemists, and from these centers have come the major contributions to our knowledge. Understandably also, these two centers have accumulated large series of cases, each numbering almost 200. Although one can garner little from the experience of the Mayo Clinic to indicate the frequency of the disease the country over, there is every evidence of the value of a high index of suspicion in making the diagnosis since forty-five cases had been diagnosed there between 1942 and 1947 in contrast to sixteen cases in the previous fifteen years [*3*]. Although it is granted that referral of patients from great distances has accounted for many of the cases seen at the Massachusetts General Hospital, it is significant that each year about five such cases are uncovered in that hospital on the house service receiving its patients in large measure from the surrounding population.

Review of the total experience with parathyroid tumors in Seattle hospitals in the eleven years from January 1, 1946 through 1956 (Table 1) reveals only eight proved clinical cases of adenoma of the parathyroids. A single case of parathyroid carcinoma will be reported elsewhere. An additional small adenoma was discovered in reviewing the microscopic studies of an autopsy on a patient whose history was devoid of symptoms of the disease. During these same eleven years the records of four additional patients were incorporated into the hospital libraries as examples of the disease. Of these, three were explored fruitlessly, two of whom from their chemical studies must surely have had the disease although neither patient was examined postmortem. Another patient improved clinically following the removal of two glands 5 mm. and 3 mm. in diameter which were histologically normal; the chemical studies were equivocal. The chemical diagnosis was certain in the fourth patient who died of renal failure at the age of eighty, too sick to permit exploration, again without the ultimate proof of necropsy. There remain, then, nine proved cases in Seattle hospitals in the past eleven years. This experience is strikingly similar to the report of the collected cases from Cincinnati by Lee, McElhinney and Gall [*19*] who during a fifteen-year period ending in 1955 found eleven cases, seven diagnosed clinically. The two communities are similar in size. In the

TABLE 1

REVIEW OF PATIENTS WITH PARATHYROID ADENOMAS SEEN IN SEATTLE HOSPITALS FROM 1946 TO 1956

Data	Race, Sex and Age (yr.)	Urinary Calculus	Bone Disease	Intrinsic Renal Disease	Adenoma Found
Swedish Hospital, 1947....	W, F, 74	No	Marked	Severe	Autopsy, right lower pole
Swedish Hospital, 1948....	W, M, 48	Many	No	No	Right lower pole behind esophagus
Virginia Mason, 1951	W, F, 35	Solitary	Marked	Mild	Right upper pole
Swedish Hospital, 1952....	W, F, 57	Solitary	Marked	No	Left lower pole
Virginia Mason, 1954.....	W, F, 61	No	Moderate	Severe	Left superior pole
Swedish Hospital, 1955....	W, M, 55	Many	No	Mild	Two adenomas, both lower poles
Swedish Hospital, 1955....	W, F, 26	Many	No	Mild	Right lower thymus, intrinsic
Swedish Hospital, 1956....	W, M, 37	Solitary	No	No	Left lower pole

Salt Lake area, likewise, Heurie, Nelson and Castleton [*16*] reported four cases proved through 1954. There is little evidence in these communities that the disease can be considered "relatively common," as both Cope and Black consider it.

A very pointed conclusion must be reached by examining the records of the Seattle cases: that the frequency with which the diagnosis of hyperparathyroidism is established bears close relationship to the index of suspicion of that diagnosis in the minds of the staff of a given hospital. (Table 1.) Thus it will be seen that of the nine cases proved in this city, six came from the surgical services of one hospital and three from another. None was uncovered in the nine other hospitals whose records were examined, although the type of practice in these (representing six private general hospitals, two Federally controlled hospitals and the King County Hospital) differs in no way significant of the frequency of this condition among their patients. It would therefore seem appropriate to re-emphasize the symptoms and signs of the disease and to suggest a routine for the survey of the patient once the diagnosis is entertained.

Briefly, the disease syndrome may be broadly described as follows: (1) Rise in the serum calcium reduces the excitability of the nerve-muscle apparatus resulting in muscular weakness, constipation, loss of appetite, nausea and vomiting, cramp-like abdominal pain, dysphagia. There is generalized muscle hypotonicity. The electrocardiographic Q-T interval is shortened. Auditory acuity suffers. Thirst and its consequent gratification occur, frequently resulting in excessive intake of milk (polygalactodypsia). (2) Calcium is deposited, earliest in the urinary tract, but also in the lungs, the arterial walls, heart muscle, gastric mucosa and even the thyroid. (3) Kidney manifestations are hyperphosphaturia and hypercalciuria, typically staghorn stones in the renal pelvis or renal calcinosis. (4) General and focal calcium release from the skeleton results in bone cysts, generalized decalcification, brown tumors, resorption of the lamina dura about the teeth and "ground glass" skull.

Involvement of the bones has been stressed for over thirty years, and those who suffer from the striking changes of osteitis fibrosis cystica, solitary cyst or epulis, as well as generalized osteomalacia, present a condition so striking that it is not surprising that their recognition up to this time has been far more likely than recognition of the relatively more common condition of renal lithiasis. Even in 1936 Cope [*9*] considered these cases with osseous manifestations "museum pieces" and pointed out that we must look for the majority of the cases in the future among patients with renal stones.

The group with neither symptomatic osseous change nor kidney stones present such esoteric complaints that they are likely to be considered functional rather than organic. The diagnosis has been established in only five cases we were able to find. Within the story told by both patients we have treated are vague muscle aches and pains, weakness, lassitude, gastrointestinal discomfort resulting in repeated search for an active ulcer (demonstrated radiographically in Case 1), social and marital maladjustment resulting in divorce in each case, histories so poignant that the mere recitation

of the difficulties resulting from the urinary tract stones fades into a minor place in the symptomatic picture of either patient. Although our observation of the second patient is brief and less complete since she lives in another community, she has achieved much greater stability as well as physical comfort from the ablation of the adenoma. In the instance of the first patient there has been an entire reversal in personality. From an antagonistic, erratic, alcoholic, surly person unable to continue his responsible position, he has reverted to a stable, humorous, frequently gay, effective executive and father. One has only to see such a metamorphosis resulting from the extirpation of a parathyroid adenoma to realize that few pathologic conditions present greater opportunity to the physician to be of service to his patient by establishing the diagnosis and to the surgeon in removing its cause.

Bone manifestations of hyperparathyroidism are more likely to appear late in the disease. Such a patient will properly come under the purview of the orthopedic surgeon and the trained radiologist, who should be quick to recognize these changes as manifestations of parathyroid hyperfunction. Further survey of the skeleton and x-rays of the mandible demonstrate the absence of the lamina dura of the teeth, leading to a study of the calcium and phosphorus metabolism. The problem of the urologist is, however, a more difficult one. The number of urinary stone formers who have parathyroid adenomas has been estimated as 5 per cent by Reifenstein. During the eleven years of this study the library of the Swedish Hospital records 1,786 patients seen with a diagnosis of stone in the urinary tract. We did not determine in how many of these serum calcium and phosphorus and urinary calcium excretion studies were made. Should we have made the diagnosis of hyperparathyroidism eighty-nine times rather than six? Surely this figure is too high, but there must be many people in our community with unsuspected parathyroid disease still under doctor's care. The conclusion cannot be escaped that a simple and effective survey adopted in all patients with urinary stones should result in much greater frequency of diagnosis of parathyroid hyperfunction.

To the physician responsible for the care of a patient with urinary stones we suggest the following procedure, which is neither too demanding of the time of the physician or the laboratory nor too expensive to the patient:

1. Routine Sulkowitch tests should be made on the urine of all patients with stones, even though the stones may be only in the bladder or prostate (urea-splitting organisms alkalinize the bladder urine, causing precipitation here). This test can be conducted accurately and easily in the office, and inexpensively in the hospital. Collection of urine for twenty-four hours is preferred but not essential. The quantitative determination of urinary calcium excretion on a measured diet has little if any advantage over the qualitative.

2. Further study of serum calcium and phosphorus levels on: (a) Patients with a positive Sulkowitch test. (b) Patients with calcium containing stone anywhere in the urinary tract. This is especially mandatory in patients with renal calcinosis and staghorn calculi in the kidney pelvis. It must be realized that serum calcium may be found at normal levels if there has been low intake of calcium in the diet, and that the patient in bed may excrete excess calcium in response to removal of stress from his skeleton. Severe renal dysfunction may cause diagnostically confusing renal rickets, cases that tax the acumen of the best informed. (c) Patients who, in addition to urinary lithiasis, present vague symptoms of weakness, muscle pains, nausea and vomiting, peptic ulcer or like dyspepsia.

3. Determinations made daily for three days may be repeated if the clinical facts make normal initial values suspect. Study of the serum or urinary phosphatase levels is necessary only if the serum calcium is elevated and the phosphorus depressed. Increased phosphatase activity indicates that calcium is being extracted from the skeleton. Radiographic studies may then be made. X-rays, to determine the manner and severity of skeletal changes, should begin with the mandible and the hands, and be expanded to include the remainder of the skeleton as indicated.

4. Serum protein determination checks the validity of the serum calcium values.

Survey of the patient conducted in this manner should entail minimum expense to the patient and no significant delay in his definitive urologic care.

There should be no occasion for the exploration of the neck and mediastinum for a tumor that will not be present. The biochemist comes

closer to the absolute in making a certain diagnosis in hyperparathyroidism than in any other condition with which we are acquainted. This has been gracefully expressed by Churchill and Cope [9], who wrote: "An 'exploratory operation' to confirm or disprove a doubtful diagnosis has little place in this field. The findings of the laboratory are more exact than the dissection of the surgeon, and there is no point in the operation at which the operator may lay down his scalpel and find comfort in having disproved the diagnosis." The surgeon searching for parathyroid disease must be convinced that the tumor has to be there before he undertakes the operation. The search for parathyroid adenoma should never be unrewarded. We are in agreement, however, that stress justifiably placed on the difficulties presented to the surgeon in the proper conduct of a search for parathyroid disease may have discouraged physicians treating patients who may have the disease from attempting to establish the diagnosis. Experience in recognition of the parathyroids may be readily accumulated by the surgeon who will look for these bodies while operating upon the thyroid and at the autopsy table. In the morgue both he and the pathologist may add to their familiarity with the frozen section diagnosis. One incident in Seattle emphasizes the value of such an exercise: In Case VII the surgeon removed what he thought was an enlarged and pathologic parathyroid which was called "normal" on frozen section examination. Having located three other smaller glands, the surgeon closed the neck discouraged only to have the correct diagnosis of adenoma returned upon examination of the fixed sections two days later. The patient was cured. The surgeon also recovered.

Development of effective means for the control of postoperative tetany has made it less imperative that no normal parathyroid tissue be sacrificed. Although we certainly do not advocate needless sacrifice of potentially normal parathyroid tissue for biopsy, it is necessary to leave only a small vascularized fragment to permit its hyperplasia to supply the patient's need for parathyroid function. We also question whether Cope's [10] admonition that the sternum should not be split to permit exploration of the anterior mediastinum at the same sitting is at present valid unless the patient be in the far advanced stages of the disease. Certainly the surgeon undertaking a search for parathyroid disease should be prepared to split the sternum and be aware of the possible locations of parathyroid tissue in that region. Having located less than four glands in the neck of a patient in good clinical condition, splitting the sternum to find the fourth and probably abnormal gland adds little to the risk and morbidity of the procedure. We are of the opinion that the staff of every hospital where good general surgery and competent pathology are practiced should be expected to handle parathyroid surgery competently.

It has happened that the two patients one of us (F. J. J.) operated upon have both had parathyroid adenomas in remote locations. Case II reinforces the opinion that total resection of the thymus is indicated if no tumor is found elsewhere. Brewer [6] has the honor of being the first to call attention to the occurrence of parathyroid bodies in the thymic substance, reporting two parathyroids within the thoracic thymus near the upper pole in adults and in heterotopic cervical thymus fragments in infants. Cope [10] has reported three cases of parathyroid adenomas lying adjacent to or within the thymic capsule, and Norris [20] reported six more cases in his collected review. Abrams, Rutenberg, Lesses and Cargill [2] reported a case in which there was a completely intrathymic parathyroid adenoma near the left lower pole. Our patient presented only a 1 mm. speck of thymic tissue on the anterior surface of the lower pole of the right thymic body overlying the pericardium. The totally buried normal parathyroid III on the left was found by the pathologist within the lower pole of the left thymus.

CASE REPORTS

CASE II. F. F., a forty-eight year old executive, was admitted to the Swedish Hospital in 1944 because of "kidney gravel." During the next three or four years he was hospitalized on four occasions in Ketchikan and during this time he passed kidney stones. He sought the aid of his urologist again in 1948 during a particularly severe attack and was admitted again to the hospital, dehydrated following three days of nausea and vomiting and pain in the left flank.

Twenty years before, he had been in the Swedish Hospital with a tenacious tracheobronchitis following an acute upper respiratory infection. Weakness, a 20 pound loss in weight, vague abdominal pains and constipation were prominent in his history. During his thirties he had been repeatedly seen by

an internist because of abdominal pain thought to be peptic ulcer, vague muscle pains, constipation and headache. X-ray examination confirmed a duodenal ulcer which responded poorly to treatment. He had become increasingly alcoholic and irascible and unable to continue his responsible job, leading

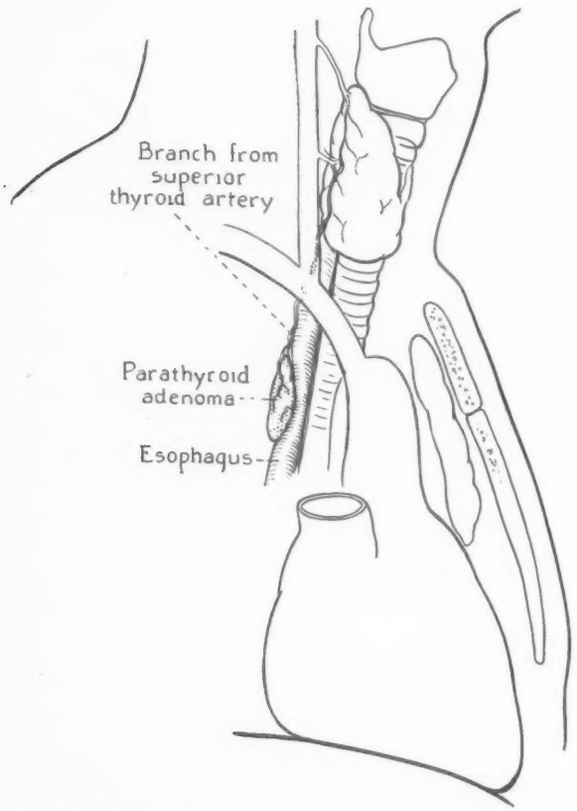

Fig. 1. Case 11. Parathyroid adenoma in the posterior mediastinum.

to the dissolution of his marriage in 1946. During a sabbatical year abroad he had remarried, and promptly upon his return to this country he again had renal colic.

In 1948, other than for the findings expected with acute renal colic, his physical examination was unremarkable. Routine blood studies were unrevealing. Blood urea nitrogen was 22.5 mg. per cent, and creatinine 1.6 mg. per cent. There were many leukocytes in the urine. No bacteria could be cultured.

Entertaining a diagnosis of hyperparathyroidism because of the recurrent bilateral calcium stones in the renal pelves with calcification of the renal parenchyma, initial serum calcium level on December 4th was found to be 10.5 mg. per cent and phosphorus 3.7 mg. per cent. Unrelenting obstruction demanded removal of the offending stone from the renal pelvis on the left.

After three days on a controlled calcium diet of 100 mg. while actively ambulant, twenty-four-hour urinary secretion of calcium was found to be 0.36 gm., 0.24 gm. and 0.29 gm. on each of three days. On the subsequent three days serum calcium values were 13.8, 15.6 and 14.3 mg. per cent; and serum phosphorus 2.5, 2.6 and 3.5 mg. per cent. Serum protein value was 6.2 mg. per cent. Radiographs of the skull, hands, legs and feet showed no abnormalities. The surgical consultant recorded a firm, rounded tumor in the left side of the neck in the region of the lower pole of the left thyroid.

The diagnosis of hyperparathyroidism established, surgery to remove the tumor was again postponed by unrelenting obstruction of the right upper ureter with pyuria and high fever, and a stone was removed from this renal pelvis.

On January 7, 1949, the neck was explored. The nodule felt preoperatively was found to be a thyroid adenoma in the left lobe, and two normal parathyroids were located on the left in the expected positions. On the right an aberrant artery and veins were noted taking off laterally from the midportion of the right thyroid lobe and were traced to a parathyroid tumor lying posterior to the esophagus deep in the posterior mediastinum (Fig. 1), typical in color and measuring 2.6 by 1.3 by 0.6 cm. The inferior parathyroid body on this side was not located.

His course was completely benign following removal of the tumor. Most striking has been his total change in personality pattern to a responsible, humorous, well integrated husband and father. He no longer complains of abdominal pains, vague aches elsewhere in his body, and is the antithesis of the hypochondriac.

Comment. Note that the initial values for serum calcium and phosphorus were well within normal limits, but that quantitative urine excretion of calcium and subsequent calcium and phosphorus levels were typically high. Location of this tumor might have been greatly aided by radiography of the cervical esophagus which was displaced to the left by the tumor. Albright's rule that the palpable tumor in the neck is in the thyroid and not in the parathyroid is borne out. It is fortunate that renal damage in this patient was not more severe after the long history, although it seems probable that correction of the condition was done just before irreversible kidney damage had occurred.

CASE VI. H. K., a twenty-six year old housewife, had her initial kidney stone in 1952. Her first visit to the Swedish Hospital was as an outpatient because of menstrual irregularities and lower abdominal pain. The fallopian tubes were found to be blocked after injection of lipiodol.

The patient's mother reported that she had had severe bilateral abdominal pains since the age of three and a goiter since childhood. She had fractured her ankle as a child. Between 1948 and 1951 she had had three children. A "chronic appendix" had been removed in 1950. She complained of feeling very tired for two years, was unable to finish jobs formerly done easily, had frequent headaches, marked constipation, inability to sleep, and was "not much good" in very hot or cold weather. She felt cold most of the time, bruised easily and her teeth were in poor condition. She had drunk 1 to 2 quarts of milk daily for two years.

Renal pain on the left developed three months before her admission of September 12, 1955, and x-rays taken by her referring physician showed a calcium stone in the left renal pelvis.

She was a pale, listless woman, her trunk obese, her extremities slender. There was generalized enlargement of the thyroid. Reflexes were hyperactive generally, but the remaining examination was unremarkable. Routine blood studies were normal. There was marked hematuria. Urinary calcium excretion for twenty-four hours on normal diet was 0.293 gm. Radiographs of the kidneys showed a 1 cm. calcification in the region of the left renal pelvis. Films of the skull and hands showed no pathologic change. A serum calcium of 13.8 mg. per cent and a phosphorus level of 1.9 mg. per cent established the diagnosis of hyperparathyroidism. The left renal stone was removed.

She was returned to her referring surgeon in another city where the neck was explored. A large parathyroid body, thought to be pathologic, was removed from the mid-position alongside the left thyroid lobe and a normal upper right parathyroid was located. Pathologic report on both of these was "normal parathyroid tissue."

She again returned to the Swedish Hospital on December 13, 1955, with a severe attack of right renal colic. Urine was remarkable for "innumerable" red blood cells and a few calcium oxalate crystals. Emergency right pyelolithotomy was performed. On a low calcium diet the serum calcium on December 19th was 14.8 mg. per cent, phosphorus 2.8 mg. per cent, alkaline phosphatase 4.0 King-Armstrong units. Twenty-four-hour urine calcium excretion was 0.299 gm.

On her third admission, on January 11, 1956, calcium values were 12.8 mg. per cent, phosphorus 2.2 mg. per cent. Surgical consultation was requested. On re-exploration of the neck no normal parathyroid tissue was visualized. The sternum was then split. The thymus was found to be bilobed, remarkably fetal in type. The left lobe of the thymus was removed and no abnormalities were noted on gross examination. On the anterior surface of the lower pole of the right lobe of the thymus overlying the pericardium there was a 1 mm. speck of tissue slightly brownish red in contrast to the pale pink of the thymic surface. Following removal the thymus was cut and within its lower pole there was an ovoid tumor 1.9 by 1.5 by 1 cm. weighing 1.7 gm. (Fig. 2.) Frozen section showed the typical histology of parathyroid adenoma. Subsequent serial sections of the left lobe of the thymus revealed a globule of normal parathyroid tissue similarly located in its lower pole.

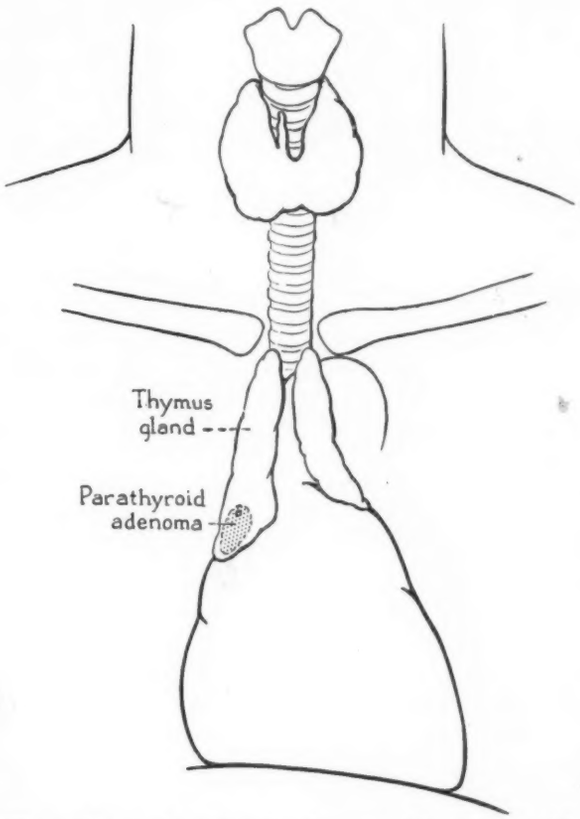

Fig. 2. Case VI. Intrathymic adenoma of the parathyroid.

Mild postoperative tetany developed which was readily controlled.

The patient's general complaints were entirely dispelled following removal of the tumor. She has resumed her place in the family and has returned to her husband.

Comment. Although it is difficult to establish the time of origin of parathyroid hyperfunction in this patient, its general effect on this woman's well-being, both psychic and physical, was severe. The inadvisability of operating upon parathyroid disease without the availability of a trained pathologist equipped to examine tissue removed by frozen section is illustrated. The tumor gave little evidence of being present within the thymic encasement and illustrates the desirability, first recognized

by Cope, of removal of both thymus lobes should no tumor be found in the anterior mediastinum.

SUMMARY

1. Hyperparathyroidism is still eluding diagnosis in Seattle and probably in similar communities.

2. General physical and psychic effects of the disease are productive of severe misery to those who suffer with it and frequently to their families.

3. The protection of a high calcium intake and wider knowledge of its skeletal manifestations make its discovery in the future much more likely in patients with urinary tract lithiasis.

4. Intractable dyspepsia suggestive of peptic ulcer or with actual peptic ulcer accompanied by weakness, polyuria, polydypsia and constipation frequently signal the disease long before precipitation of calcium salts brings on renal or ureteral symptoms.

5. Wider use of the Sulkowitch test to initiate a simple survey will result in much more frequent diagnosis.

6. Serum calcium and phosphorus levels should be determined on several days in suspected cases, since these levels may be normal in some phases of the disease.

7. A positive chemical diagnosis makes it imperative that the surgeon find the offending diseased gland or glands. Skills necessary to establish the diagnosis in the laboratory, to prove it during surgery and to confirm it in the pathologic laboratory should be available in all of the larger general hospitals.

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DISCUSSION

A. C. Pattison (Pasadena, Calif.): I welcomed the opportunity to discuss Dr. Jarvis' paper for I knew, as it certainly proved to be, that it would be an excellent one, and also it would give me the opportunity to report one of those fantastically large-in-number series of cases from Southern California. My search for cases, however, turned up rather surprising results.

At the Huntington Memorial Hospital in Pasadena since 1933 there has been but one parathyroid adenoma removed surgically. In the past ten years at the Los Angeles County Hospital there have been only four patients who have had parathyroid adenomas excised.

Your obvious response is that our index of suspicion is low and that we are not adept at diagnos-

ing this disease. However, during this same period at the Huntington Hospital (1933 to the present) no cases of parathyroid adenoma have been found at autopsy. To prove that, parathyroid glands are examined. Four cases have been encountered of hyperplasia of the parathyroids secondary to chronic glomerular nephritis. Also, at the Los Angeles County Hospital since 1918, during which period there have been over 56,000 autopsies, only six parathyroid adenomas have been found.

Most of you have read Leon Goldman's paper on "Unusual Manifestations of Parathyroid Adenomas," so I would rather not discuss the various aspects of this disease. The differential diagnosis, however, is occasionally not too simple, although all agree that the clinical diagnosis of parathyroid adenomas must be made in the laboratory. Hypercalcemia may also be observed in patients with intoxication from large doses of vitamin D, osteolytic carcinomatosis, multiple myeloma, rapid skeletal rarefaction particularly in young people with poliomyelitis and fractures, sarcoidosis and other diseases. It is essential to differentiate these from the parathyroid adenoma which is the only disease amenable to surgical treatment. The foregoing diseases may also have an associated low serum phosphorus. Also, it has been pointed out by Albright that the renal insufficiency often accompanying hyperparathyroidism frequently restricts phosphorus excretion; therefore the serum phosphorus in hyperparathyroidism with renal insufficiency may not only be normal, but also abnormally high.

Although I have had only one experience with the maneuver, Howard has reported that the spinal fluid calcium (normal values, 4.5 to 5.5 mg./100 cc.) is invariable high in patients with hypercalcemia other than that due to hyperparathyroidism, whereas the highest reported spinal fluid calcium in a patient with parathyroid adenoma has been 6 mg./100 cc. This may be a valuable differential finding and I wonder if Dr. Jarvis can add to or subtract from this experience.

Dr. Jarvis has rightfully stressed the goal of early diagnosis which should, if possible, be attained before the development of bone lesions, renal calculi or nephrocalcinosis. To illustrate this point further I would like to quote Dr. Rienhoff's study of the late follow-up in a series of twenty-five cases. There were nine late deaths, occurring three to eleven years after operation, and with one exception all these patients were in the fourth and fifth decades of life. All the deaths were due to hypertension with or without associated renal insufficiency. At the time of operation five of these patients had associated skeletal disease, one both renal and skeletal complications and four renal complications only. Our small autopsy series at the County Hospital substantiates these observations. To my knowledge, a late follow-up study on patients having parathyroid adenomas, but without complications, has not been done.

JOEL W. BAKER (Seattle, Wash.): I think that Dr. Jarvis and his associates served a real purpose when they first presented their report to the Seattle Surgical Society and appealed to our civic pride to see if we could not discover more instances of this disease. Actually I think that this paper should be heard by general physicians, internists, orthopedists and urologists as well as by us.

I would like to emphasize that while the diagnosis is made more often by the orthopedist or the urologist, the patient who happens to be given a high calcium diet may thereby have the bone changes delayed. The internist and general physician should suspect the disease in those patients who have migratory neuromuscular symptoms, unexplained gastrointestinal symptoms and even in some patients with personality changes. Recently a laboratory test has been made available to us which is much more helpful and more specific than the Sulkowitch or other tests available in the past. I refer to the renal phosphorus tubular reabsorption test. In the past year this test in our laboratory has proved dependable and has been useful in running down suspect cases and in screening patients with urinary stones.

It has been said that the bone changes indicate the duration of the disease and the renal changes indicate its severity. Certain it is that the degree of pyelonephritis and particularly the extent of tubular calcinosis determine the irreversible extent of the disease. In such advanced cases, although the surgeon may find and remove the parathyroid tumor, the longevity of the patient is still limited. He will succumb ultimately to his renal disease.

This is a disease in which the laboratory tests rule supreme in making the final diagnosis. As Dr. Jarvis has mentioned, we should not enter the neck until the diagnosis is irrefutable, and then we should continue our search until we find the offending parathyroid tumor or hyperplasia. In the past few months we have had three patients under study at the same time and in one we found a tumor. The other two patients have not been operated upon but are still under study because all laboratory tests have not been confirmatory. At operation, meticulous care in maintaining a bloodless field, division of the ribbon muscles and persisting in the search on one side of the neck until at least the normal parathyroid glands are identified are important essentials in the technic. The inferior parathyroid gland on either side is, of course, more easily demonstrated than the superior gland and fortunately is the one statistically more often involved. The small artery which supplies the parathyroid gland is the key to the search. When the search has to be carried to the opposite side of the

neck, the glands will usually be found in a symmetric position. Fortunately, in our cases the tumor was found in the neck and the mediastinum did not have to be opened. One should be prepared, however, for this contingency. I wish to thank the essayists for including our cases in their report.

LEON GOLDMAN (San Francisco, Calif.): I feel quite certain, because of similar experiences of our own, that in Seattle there will be an upsurge in the number of patients discovered to have this disease.

We feel after study of these patients that there have been some changes in the clinical picture and also in the methods which we now utilize to make the diagnosis of hyperparathyroidism.

In the first place, following Mandl's removal of a parathyroid adenoma (about thirty-two years ago) in a patient with generalized osteitis fibrosa, it was assumed that these bony changes are the prerequisite for making the diagnosis.

Nowadays less than one-fourth of the patients have osseous changes. Most of them (80 per cent) have either renal stones, calcifications or symptoms of parathyroid hyperfunction.

I would like to relate some of our experiences, which may be of value to some, and also to present a test which has helped us in making the diagnosis of hyperparathyroidism.

The classic chemical findings are, as we all know, hypercalcemia, hypophosphatemia and hypercalciuria. Since 1935 we have seen thirty-six patients with hyperparathyroidism, twenty-four of which we encountered in the last two years. All these cases were correctly diagnosed, all the patients were operated upon and all had hyperfunctioning parathyroid tissue removed.

There are several things which account for the recent upsurge in the number of patients with hyperparathyroidism. The main thing is, I believe, a sharpness in the methods of diagnosis.

With the passing of time we see fewer patients with markedly elevated calcium levels in the blood stream. Many of the patients that we have seen during the last few years have serum calcium levels under 12 mg. %. If we are to have a laboratory test to determine the level of serum calcium, it must be extremely accurate because in some patients ½ mg. is the degree of elevation.

We have also seen a group of patients having normal phosphorus levels in the blood stream with hyperparathyroidism, and who have had either parathyroid adenoma or primary hyperplastic tissue removed.

Of the last twenty-four patients seen during this two-year period, twenty-two were known to be uremic. Of these twenty-two patients, fourteen had a normal serum phosphorus level.

Dr. Fuller Albright, who actually was the leader in this country and in the world in establishing the chemical criteria for hyperparathyroidism, said in 1929 that one of the prime functions of parathyroid hormone was the increased excretion of phosphorus by the kidney. Later, other investigators showed that the prime function was actually the tubular reabsorption of phosphorus from the tubule into the blood stream; and that if there were a method of measuring the tubular reabsorption of phosphorus, we would then have an accurate index of parathyroid hyperfunction.

Dr. Gilbert Gordan of our Medical Department has been interested in this test. We have applied it during the past two years and it has not failed us as far as reaching a diagnosis is concerned. It is a simple test to do and one which I would recommend, because the calcium level is changed so little in some of these cases. Also, as I have said, we have seen some patients with normal phosphorus levels and this test helps us to make a decision in the borderline case.

The excretion of phosphorus by the glomerulus is actually one of filtration; and as the phosphate passes down the tubule, about 85 per cent of it is reabsorbed in the normal person. If we can measure this, we have quite an accurate index of parathyroid hyperfunction.

In one series of thirteen normal persons, TRP (tubular reabsorption of phosphorus) determinations averaged 84.7.

In patients with osteoporosis (which in many cases was postmenopausal osteoporosis) there was a normal tubular reabsorption of phosphorus of 83.7.

In patients with renal stones without hyperparathyroidism there was a normal reading of 84.

In ten patients with hyperparathyroidism the reabsorption was low, indicating a loss of phosphorus through the urine. In this series the average reading (TRP) was 65.

In a group of patients in whom the diagnosis of hyperparathyroidism was made, the patients were operated upon and hyperfunctional tissue was removed. The preoperative TRP determinations were all low, the average being around 85. We have had TRP readings varying from 51 to 78 in our series. As in every other test, there is a border line zone between the normal and the abnormal; 78 and 77 are getting close to normal.

Immediately after the removal of the hyperfunctioning tissue the TRP reading goes back to normal for many days; as long as these patients are tested, their TRP levels return to normal.

This has been also of some interest to us because we have made mistakes in overlooking hyperfunctioning parathyroid tissue. We had one patient with primary hyperplasia. We removed four parathyroid glands, all of which were very large, and we left a small part of one. The TRP determinations were still low postoperatively, and we know that this patient has a fifth or possibly sixth parathyroid mass.

Fuller Albright said many years ago that if a patient with hyperparathyroidism has enough phosphorus in his diet, the phosphorus level will go up to normal; if the calcium intake is not very high, the calcium level may drop so that one will then miss the diagnosis.

Reifenstein suggested to us that although we had some patients with a normal phosphorus level, the normal phosphorus level in the blood would drop below normal if we put these patients on a restricted phosphorus intake.

We did that with these patients; the phosphorus level in the blood did drop and we actually produced hypophosphatemia. The TRP reading in these patients was low, which indicated hyperparathyroidism.

One normal person had a phosphorus level of 3½ mg. per cent; there was a restricted intake of phosphorus in the diet and a reabsorption of about 85 per cent. A certain amount spills over into the urine.

In the patient on an "American" diet (which contains plenty of calcium and usually plenty of phosphorus) there is a low spilling over of TRP. However, he is taking so much phosphorus in the diet that his blood level stays up to within normal limits.

In this series of twenty-two patients, fourteen had normal phosphorus levels. We went back over the records of some of the patients studied previously whom we thought at that time did not have hyperparathyroidism. We now applied the phosphorus deprivation test to these patients and showed that the phosphorus level would come down under those conditions. We then operated upon them and found hyperfunctioning tissue.

We should be aware that the chemical changes, as far as calcium and phosphorus are concerned, can be at inconspicuous levels and yet the diagnosis can be made. The TRP test is a valuable aid. It is a simple test which can be done using one blood specimen and one untimed urine specimen, and does not annoy the patient. It is important to remember that patients with hyperparathyroidism may have normal phosphorus levels; this may escape our observation if attention is not paid to the patient's dietary intake.

CLARENCE J. BERNE (Los Angeles, Calif.): I, also, wish to indicate how much I enjoyed Dr. Jarvis' paper. I personally, for a long time, have had considerable interest in this disease because I believe that I was the first assistant at the second parathyroid tumor removal in this country. Dr. Jarvis may straighten me out on that.

I would like to report briefly on two cases that call attention to some of the peculiarities which come up in meeting the problem of hyperparathyroidism.

The first is that of a woman thirty years of age, who was admitted to the hospital in a coma. She had had marked polyuria for about four weeks. When she was admitted, she was hypotensive and oliguric. It was possible to bring her blood pressure up to normal. It was ascertained that she had hypercalcemia and the other classic laboratory findings of hyperparathyroidism.

The patient's course had been extremely short. She began vomiting, and I think her coma was accelerated by the vomiting and the hypovolemia that developed.

We explored the patient's neck under local anesthesia while she was in coma, and removed a malignant parathyroid tumor. Interestingly enough, the patient's blood calcium did not change subsequently. This was, I believe, due to the patient's lack of renal function and she died about six days later because of acute renal insufficiency

I believe this is an interesting sidelight into the fact that the feed-back mechanism in the bone probably was not adequate to reduce the patient's hypercalcemia, the reduction of which actually depended on renal function. She had no renal function, and at autopsy extensive tubular necrosis with severe metastatic calcification was seen. I suspect that the terminal acute renal insufficiency was due to shock secondary to dehydration dependent on the patient's basic disease.

Therefore she is categorized, I think, as having had a hyperhyperparathyroidism or so-called acute hyperparathyroidism. The patient's entire course was highly fulminating and her tumor was malignant.

What I would like to call attention to particularly is that the patient was admitted to the hospital in coma due to hypercalcemia.

The second patient was also admitted in coma. This patient had a history of severe duodenal ulcer. He had been drinking a great deal of sour milk, and it was ascertained ultimately that his coma was the coma of hypercalcemia; that his basic problem was Burnett's syndrome, and that the problem was dependent on high calcium ingestion. Nevertheless the hypercalcemia had produced a coma similar to that of the woman previously mentioned.

Burnett's syndrome is well established. It is not clear, however, to all of us that the syndrome may cause coma.

Another interesting fact about this man is that he had a duodenal ulcer, and the allusion has been made earlier in the meeting, I think it is generally said that duodenal ulcer occurs eleven times as often in patients with hyperparathyroidism as in the general population.

This man was treated on a reduced calcium intake, and then within six or eight months had a spontaneous compression fracture of D-10 and L-1. He was then restudied on an Aub diet. The normal excretion on an Aub diet should be less than 100 mg. per day. He consistently had 250 to 300 mg. output.

We ultimately explored this man's neck and could find no parathyroid tumor; we removed one parathyroid, and frozen section on it showed no atrophy. We therefore assumed that he did not have a parathyroid tumor and therefore removed a second normal parathyroid.

Subsequent to the operation the man has shown, on an Aub diet, an excretion well below 100 mg. per day, so that his high calcium excretion has been markedly reduced by removing two normal parathyroids.

I realize that there are objections to some of the thinking on this patient's case. However, I think it is fair to say we could all see that this patient (an older man) had osteoporosis and his basic problem was one of bone thinning; also, that parathyroidectomy of the normal parathyroid should not have been performed, perhaps, but that it would not have made any difference.

The patient is much improved so far as his general health is concerned, especially in the disappearance of his bone pain. He is now not excreting excessive quantities of calcium and seems, metabolically, to be where one would like to have him. His initial syndrome was coma due to hypercalcemia on the basis of the Burnett syndrome rather than of hyperparathyroidism.

H. Rocke Robertson (Vancouver, B. C.): I hesitate to discuss this with my small experience, particularly compared to Dr. Goldman's, but I would like to confirm two of the points made.

I can confirm, first of all, that these cases will not be found unless somebody is looking for them particularly. I believe that we had only two cases found in the ten-year period since the war, until about a year ago.

The second point that I can confirm is that they will surely be found if somebody does bother to look for them.

About a year ago we started to recall patients who had recurrent renal calculi, and investigate them in our metabolic unit. To date we have only recalled some forty-five cases; but as Oliver Cope and others would have predicted, we have noted that in four of these patients chemical tests have suggested that they had tumors, and in two of the patients who have been explored tumors were found. Thus we, too, like many others, have been harboring these cases for many years without recognizing them.

I have wondered what has happened to these people that we have been missing over the years. We, like Dr. Pattison, have not found them at autopsy. Hyperparathyroidism is certainly a real disease; it definitely can kill people, and there must be many in whom the development of the disease is very slow and unimpressive.

Fred J. Jarvis (closing): I cannot add anything further here except to say, in answer to Dr. Pattison's question, that I know nothing about the relative levels of the spinal fluid calcium, but perhaps Dr. Goldman could elucidate upon it. I am especially pleased that the tubular reabsorption test has been presented so beautifully by Dr. Goldman, in view of the fact that in Seattle, as yet two "star" physiologic chemists from Dr. Fuller Albright's laboratory who are new in our community have not been very encouraging about it. We will watch with a great deal of interest.

Again, I think that the message we carry is that this disease needs a great deal more propaganda in our home communities.

Current Investigation in the Homograft Problem

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As a problem in basic surgical research the existence of tissue incompatibility to grafting between individuals of the same species is unique. On the one hand it appears to manifest obstacles which may be insurmountable, yet on the other hand the potential which exists should the problem be solved is so great that many aspects of medicine as well as surgery would be revolutionized.

It is important to note that even though in general terms the homograft problem remains unsolved, nevertheless, limited applications of a practical nature already exist. Transplantation of homostatic or non-living tissue is already widely practiced and has marked a new era in the surgery of arteries. Homostatic tissue transplantation also has a limited use in surgery of the dura mater, bone, tendon and fascia.

Intermediate in position is the success which has been established in corneal homotransplantation, in that a relatively acellular but nevertheless probably viable or homovital substance is successfully transplanted with a noteworthy incidence of success. True homovital transplantation of cellular tissue in the human being is possible only in the case of blood transfusions and, very questionably, in rare instances when successful grafting of fetal tissue has been claimed [*1*].

In the field of experimental zoology the transplantation of tissue from one embryo to another has long been practiced with remarkable success by embryologists. Interchange of tissue between infant individuals has a definite incidence of success in the chicken and probably other species. Homovital grafting between unaltered adult individuals is possible only between identical or monozygotic twins; in this situation, however, tissue compatibility is complete and permanent.

INITIAL WORK

Our initial efforts in the tissue transplant laboratory at the University of California Medical Center, Los Angeles, involved the transplantation of limb buds between seventy-two hour old chick embryos with the purpose in mind of attempting to hatch both host and donor so that various regrafting experiments involving skin from the transplanted limb could later be performed. In the course of many hundreds of operations we were successful in hatching a few "three-winged" chicks which grew into adults. (Fig. 1.) Viability and normal growth of the transplanted limb occurred with no evidence of a homograft reaction. If loss of the limb occurred, it appeared to be the result of circulatory impairment from insecure attachment of the limb and the host body at the time of the original operation. Unfortunately, we were never able to hatch a donor and found that the over-all mortality in such experiments was in the vicinity of 95 per cent. These experiments were consequently abandoned in search of a more fruitful approach to the problem.

We noted that in 1929 Danforth and Foster [*2*] of Stanford reported a definite though low incidence of permanent healthy survival of skin homografts interchanged between day old chicks. We first repeated this experiment using four pairs of day old chicks. We were fortunate to obtain permanent homograft survival in both members of one pair, a situation almost never

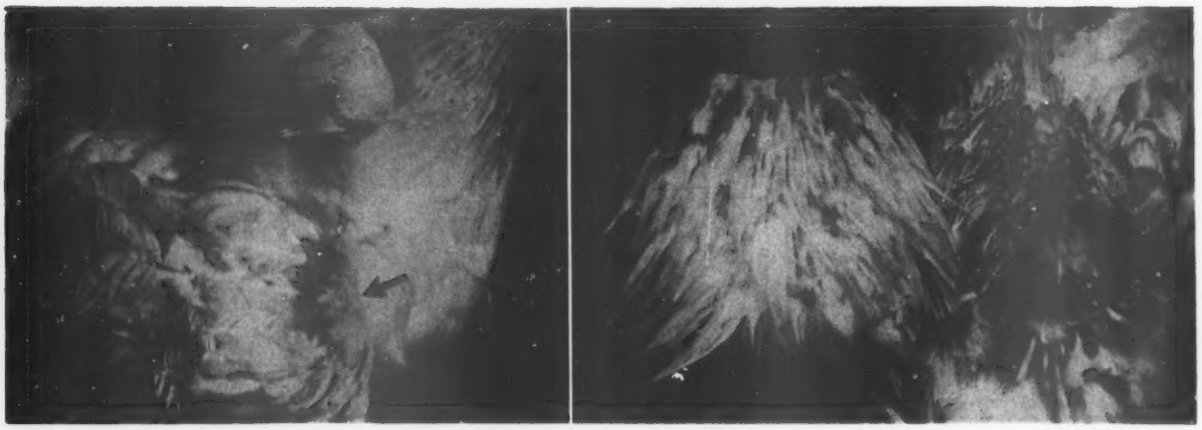

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FIG. 1. Three-winged adult chicken produced by transplantation of limb bud from embryonic donor to embryonic host at seventy-two hours of incubation. Arrow indicates wing resulting from the limb bud transplant.

FIG. 2. Homografted, eleven month old pair of adult chickens. Each member bears a healthy adult homograft produced by interchange of 1 cm. skin squares between the members of the pair at one day of age by the method of Danforth and Foster.

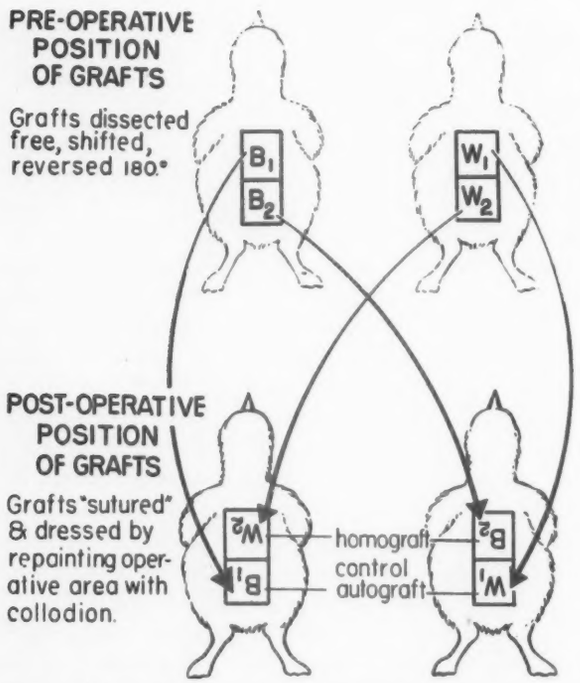

FIG. 3. Method of producing skin homografts with control autografts in young chick pairs. Diagram shows standardized method of graft transfer so that each postoperative chick bears a reversed homograft and autograft. (From: CANNON, J. A. and LONGMIRE, W. P., JR. *Ann. Surg.*, 135: 60, 1952.)

since encountered. (Fig. 2.) Stimulated by this fortuitous result, we developed a technic of homografting day old chicks which permitted operation on large numbers of chicks in a short time and included the use of control autografts. (Fig. 3.) The method consisted of placing a 1 cm. skin homograft and skin autograft on the back of each chick in a reversed position and holding the grafts in place with a coating of flexible collodion which served admirably as both suture and dressing for the grafts. By this method as many as fifty chicks could be grafted in a two-hour period. The resulting incidence of homograft take or reaction could then be plotted with statistical significance.

The behavior of skin homografts in day old chicks was then subjected to extensive scrutiny by this method. It was first noted that the more closely related were the two breeds of chicks used, the higher was the incidence of permanent homograft takes. (Fig. 4.) Nevertheless, homografting between day old chicks of any two common breeds rarely resulted in a permanent homograft take of less than 5 per cent. It was noted that an incidence of homograft survival occurred only in chicks grafted before reaching seven days of age. Chick pairs grafted when more than seven days old, though showing usually a 100 per cent take of autografts, never showed homograft survival longer than two to three weeks at most. On the other hand, if skin from chicks of increasing age was grafted onto day old chicks, a definite though decreasing incidence of long term homograft survival was obtained until the skin used was fourteen or more days older, at which point the grafts were uniformly sloughed. Split-thickness grafts of adult skin placed on day old

Current Investigation in Homograft Problem

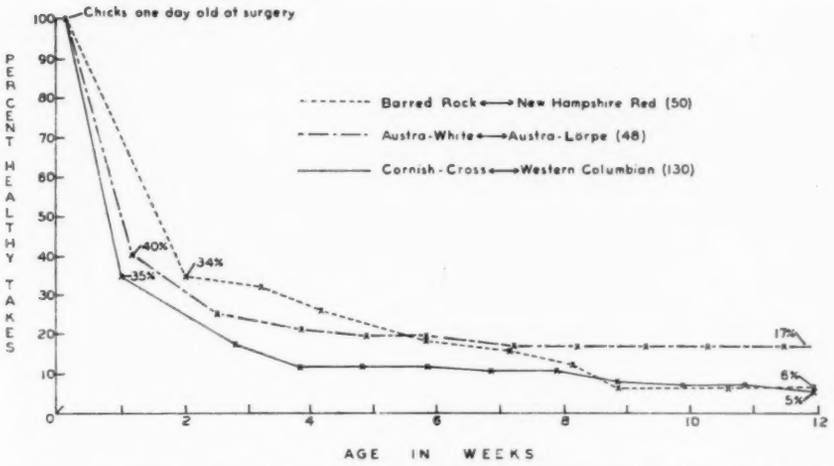

FIG. 4. Curves obtained by interchange of skin homografts between chick groups of various breeds. The Austra-White↔Austra-Lorpe curve shows the highest incidence of homograft survival. These two breeds are very closely related.

chicks were invariably rejected promptly. If skin from day old chicks was placed on chick hosts of increasing age, the day old skin would invariably be sloughed by chick hosts seven days of age or older. (Fig. 5.)

In the course of such experiments the original aim of the initial research was accomplished, in that a stock of adult host-donor pairs was accumulated with one member of the pair bearing a healthy homograft that showed normal growth and adult feathering. (Fig. 6.) With such preparations it was then possible to study the response of the homograft to certain regrafting experiments. Considerable technical difficulty was, and still is, encountered in the grafting of split-thickness adult chicken skin which made interpretation of results difficult. However, on the basis of numerous such experiments, certain impressions were obtained. If a split-thickness healthy adult homograft is returned to its original owner, a take of this graft does not occur more than 50 per cent of the time. If a repeat homograft is performed between original donor and original host, a take of the repeat homograft is almost never obtained in spite of frequent, continued health and viability in the original but now adult homograft. In this situation a reaction followed by recovery in the original homograft has been observed during the period of complete rejection of the repeat homograft. We have not observed simultaneous complete rejection of the original homograft at the time the repeat homograft is rejected.

ACQUIRED TOLERANCE

Highly fruitful and provocative results in this problem along another avenue of research were initiated by Billingham et al. [4] on observations of the response of dizygotic or fraternal twin calves to the interchange of skin homografts. In this situation where the certain dizygotic derivation of the twins is obvious by their being of opposite sexes, the skin homografts are usually permanently tolerated and behave exactly as autografts. Owen [5] showed that such twin calves each possess red cells of two types, their own and those of their twin, i.e., red cell "chimerism" exists between them. Owen further pointed out that in cattle complete placental blood vessel anastomosis develops, and hence circulatory interchange or embryonic parabiosis always takes place when fraternal twinning occurs. Billingham, Brent and Medawar [6] were then successful in experimentally inducing tolerance to homografts by injecting blood or cells of the future homograft donor into embryonic animals. This phenomenon led to the development of the theory of immunologic tolerance to homografts, which, in brief, is as follows: If living cellular material is injected from a donor into a host in an embryonic or in some cases infant stage, the homograft response is in some way altered so that a later challenge of the host with a skin graft from the donor of the injected cells is tolerated—sometimes permanently and at least usually for a much longer time than would occur in the absence of pretreatment. The

FIG. 5. Ten month old adult chicken bearing a healthy, reversed homograft and autograft. Grafts were applied when host and donor were one day of age.

exact manner in which this mechanism operates remains to be explained.

RECENT WORK

Our recent work has involved a more detailed study of the phenomenon of acquired tolerance in the chicken than has been previously accomplished. In addition, we have begun comparison studies of what we have come to call the Danforth preparation, the subject of our earlier investigations, and the Billingham preparation, just described. Distinct differences have been noted.

Through our combined ideas and discussions, but largely through the technical efforts of one of us (P. I. T.), a technic has been developed which allows the cross injection of blood between large numbers of chick embryos in a short operative period with low mortality and normal hatch rate. (Fig. 6.) The result has been groups of chick pairs in which some degree of tolerance to skin homografts between each pair has been induced. These individuals can then be studied by our method of rapid skin homografting. To date we have studied the following factors:

Age of Individual at Time of Cross Transfusion. Groups of eggs, ten to eleven and twelve to eighteen days of incubation, and chicks, one to three days old, were subjected to interchange of 0.2 to 1.0 ml. of blood. Because of our past studies which indicated that skin homograft rejection is universal in chicks by the fourteenth day of post-hatching, we chose to perform the test skin homografts at this age. The grafted chicks were then maintained until the homografts had sloughed or until the chicks were at least four months postgraft. Results of this experiment are shown in Figure 7. Blood in the amount interchanged at any age induced tolerance in only a variable fraction of the individuals treated. The highest incidence of long term graft survival occurred in chicks treated at twelve to eighteen days of incubation. The lowest incidence was seen when the interchange was effected between newly hatched chicks. Results in the latter group were characterized by a delayed profound drop in the incidence of healthy homografts, from 40 per cent to 4 per cent beginning at approximately the eighth week postgrafting.

Age of the Individual at the Time of Grafting. In this experiment chick embryos at ten to sixteen days of incubation were subjected to cross transfusion of 0.2 to 0.4 ml. of blood. Skin homografts were then performed at two days post-hatching in one group and at fifteen days post-hatching in the other group. In the first group an initial 90 per cent take of the homograft gradually dropped off to an incidence of 37 per cent at thirty-two weeks postgrafting. In the second group an initial incidence of 60 per cent dropped off to 25 per cent at the end of thirty weeks. (Fig. 8.)

Experiments Involving Repeat Homografts between the Same Host-Donor Pairs. These experiments were designed in an attempt to demonstrate the possible existence of both host tolerance and graft adaptation as mechanisms involved in homograft acceptance.

Simple interchange of skin grafts was first performed between fifty-four pairs of untreated two day old chicks. When they reached fifteen days of age, a second skin homograft was interchanged between each pair. In this group it was found that the survival of the primary or two-

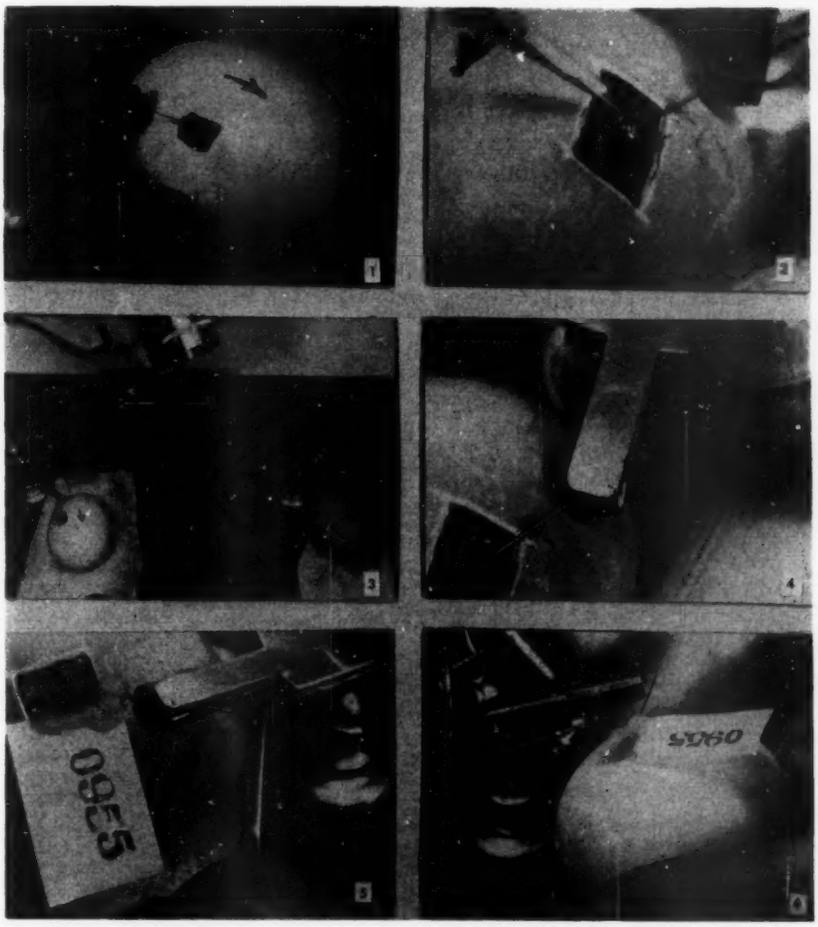

FIG. 6. Steps in technic of cross transfusion of blood between chick embryos to produce later incidence of homograft tolerance. (From: TERASAKI, P. *Proc. Soc. Exper. Biol. & Med.*, 94: 73, 1957.)

day homografts was reduced to zero from an expected 5 per cent incidence, whereas the secondary or fifteen-day homografts, although they all sloughed, appeared to remain viable for a slightly longer period than did the controls. (Fig. 9.)

A similar though more obvious result was encountered when the same experiment was performed on a group of twenty chicks which had been subjected to cross transfusion of 0.2 to 0.4 ml. of blood at ten to sixteen days of incubation before being homografted at two and fifteen days. Compared to transfused controls grafted at two or fifteen days, the two-day or primary grafts showed a decreased incidence of permanent survival. The secondary or fifteen-day grafts showed an initial slight increase in take as compared to controls with a final decreased permanent incidence. (Fig. 10.) It is, we believe, important to point out that in this experiment the secondary graft on any given individual invariably sloughed before the primary graft, and that there was a very significant incidence of primary homograft survival in spite of secondary homograft slough. This situation is analogous to that obtained in the original adult regrafting experiments on the Danforth-type preparations.

COMMENTS

In the eventual solution of the homograft problem the answers to two basic questions would appear to be of vital importance. First, what is the exact mechanism of this reaction? Second, is tissue specificity an alterable characteristic or one which is inalterably fixed by the genes and chromosomes in the nuclei of every cell of the individual? It would appear that progress is steadily being made toward the answers to both of these questions, although

the work is in the stage where data are stili being collected and interpretation of the data has barely begun.

That the homograft reaction involves an immunochemical mechanism was suggested early by Holman [9] and is fairly well supported

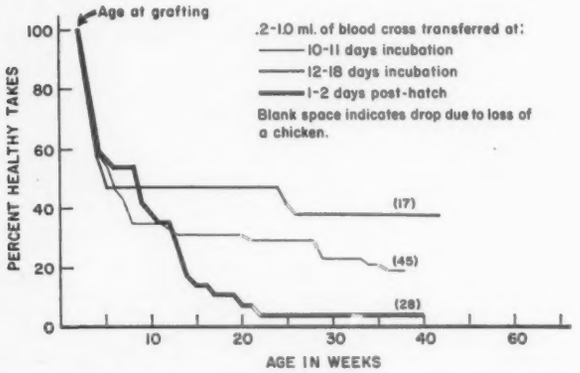

FIG. 7. Effect of age at which blood cross transferred. Incidence of homograft takes in groups of chicks cross transfused at various incubation times. A clear space in a graph line indicates a drop in the percentage curve occasioned by a mortality in the group. Optimum age for cross transfusion by this method appears to be at twelve to eighteen days of incubation.

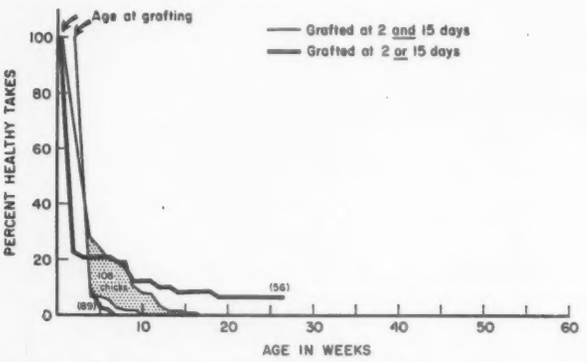

FIG. 9. Effect of grafting at two and/or fifteen days on untreated chicks. See text for discussion of this experiment.

by the data of Medawar et al. [10]. Second set skin grafts in rabbits are rejected much more rapidly than first set skin grafts from the same donor. Second set reactions to skin from a given donor can be induced in a second host by injection of lymph node tissue from a host who has reacted to a homograft from the same donor. This same second set phenomenon can be induced by injection of extracts of donor cell nuclei prepared by ultrasonic breakdown. The donor cell nuclear extract is injected into the host animal. After a suitable period, challenge of this animal with a skin homograft with skin that is isogenetic with the donor of the nuclear extract results in a typical accelerated second set reaction in the graft. The substance involved appears to be a desoxyribonucleoprotein [10].

We readily accept the experimental data rela-

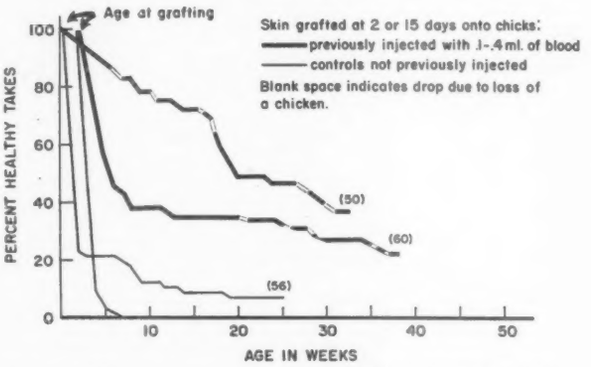
Age at grafting
Skin grafted at 2 or 15 days onto chicks:
previously injected with .1-.4 ml. of blood
controls not previously injected
Blank space indicates drop due to loss of a chicken.
PERCENT HEALTHY TAKES
AGE IN WEEKS
(50)
(60)
(56)

FIG. 8. Effect of age at which chicks are grafted. See text for discussion. A clear space in a graph line indicates a drop in the percentage curve occasioned by a mortality in the group.

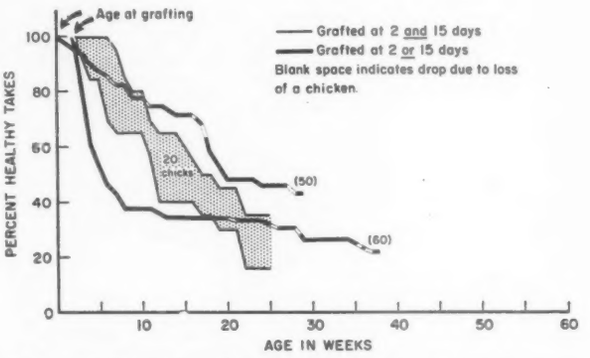
Age at grafting
Grafted at 2 and 15 days
Grafted at 2 or 15 days
Blank space indicates drop due to loss of a chicken.
PERCENT HEALTHY TAKES
AGE IN WEEKS
(50)
(60)

FIG. 10. Effect of grafting at two and/or fifteen days on chicks previously injected with blood from skin donor. See text for discussion. A clear space in a graph line indicates a drop in the percentage curve occasioned by a mortality in the group.

tive to the phenomen of acquired tolerance which are accumulating; in the light of our own experience, however, our present explanation of certain aspects of the total data differs from that of Billingham and co-workers. Contrary to Billingham, our data continue to prompt us to consider the possibility that graft adaptation, and hence alteration of individual tissue specificity, may occur in the chicken under certain of the experimental circumstances we have imposed. One experiment which might be interpreted as lending support to this concept is seen in the situation in which a host tolerates a homograft from a given donor

when the graft is applied when host and donor are one day of age; but when challenged from the same donor when both are older, i.e., two weeks or adult, the secondary homograft may be rejected while the primary graft survives. The fact that we succeeded in returning only 50 per cent of our adult homografts to the original host with graft survival would appear also to be evidence in favor of this concept, although technical problems have made accurate interpretation of this particular aspect of the work difficult. In addition, it would appear to us that our data may indicate that in the chicken not only do changes take place in the host-reacting mechanism during the first few days after hatching, but also that maturational changes in the tissue specificity of homografted skin, which is from one to fourteen days of age, appear to occur. Finally, the fact that twin calves and indeed chickens subjected to embryonic parabiosis by the method of Hasek [*12*] are altered in such a way as to manifest permanent tolerance to homografts between the pairs would seem to belie an inalterable genetic linkage for tissue specificity of the individual regardless of the mechanism involved.

The tentative concept of one of us (J. A. C.) with regard to the nature of the total mechanism is as follows: (1) Tissue specificity may be a function of the immune mechanism of the host. (2) An antigen-antibody mechanism may be involved, but cellular contact between the lymphocytes of the host and the antigenic homograft is required for the reaction to be initiated and completed. (3) It is possible that in the embryo individual specificity within the species before the individual's immune mechanism is developed does not exist. Certainly it does not exist between sperm and ovum. (4) The developing immune mechanism may well determine not only the reactive sensitivity of the host to foreign cells, but also the individual specificity of its cells. (5) More specifically, at the time that the immune mechanism of the embryo develops, the developing lymphatic system may elaborate a protein antigen of a highly specific nature which is imparted to the nuclei of the individual's tissues and is thereafter potentiated as the individual's tissue specific antigen. The immune mechanism elaborating this antigen will from then on, if challenged, elaborate antibodies within its lymphocytes against tissue specific antigens of any other individual's tissues but not its own. (6) The nature of the tissue specificity which becomes fixed in the cells of the individual upon the maturation of its immunologic system may be determined by factors presented to the developing immune mechanism which have obviously developed through gene control. However, if this individual's immune mechanism is presented with the same group of specific factors by administration of cells from a donor before immune maturation in the host has occurred, these tissue specific factors may be added to the total pattern of the individual so that future tolerance of donor tissue by the host then exists, because host and homograft share tissue specific antigens which control initiation of antibody reaction in the host's immune system.

A crucial experiment for the foregoing concept will still involve embryonic limb bud grafts and hatching of both host and donor. If skin from the grafted limb were rejected invariably by the original donor once host and donor became adults, considerable credence would be added to the foregoing concept, since undeniable alteration of tissue specificity could be claimed for the skin of the grafted limb.

In all the work heretofore alluded to, tissue compatibility has been accomplished only by the use of embryonic or infant host individuals in the initial part of the process. In no instance does the unaltered adult accept unaltered homograft unless genetic identicalness exists. Production of homograft tolerance between adults to date can be accomplished only by extensive and drastic destruction of the host immune mechanism by total body radiation followed by immediate injection of marrow cells from the donor and, later, test skin homografting with skin isogenetic with that of the donor of the marrow. The work of Main and Prehn [*13*] and Trentin [*14*] shows that adult mice so treated will tolerate such skin homografts indefinitely.

It is obvious that none of the work to date would permit contemplation of clinical applications, although certain aspects of the radiation experiment mentioned heretofore are provocative of certain limited clinical considerations.

At present, human kidney homografts have been tolerated by the host as long as five and a half months [*15*]. The resulting palliation in patients otherwise doomed to uremic death was dramatic even though temporary. Compared to the results presently obtained in many palliative cancer operations, even three months

of efficient palliation afforded by a renal homograft to a patient dying of uremia may be worthy of consideration. If by some means an average palliation of one year could be offered such patients, it would seem proper to offer the procedure.

SUMMARY

The present status of the homograft problem from a practical aspect is briefly reviewed. The initial work in the tissue transplant laboratory at the University of California Medical Center is summarized. Previous work on the phenomenon of acquired tolerance is briefly reviewed. Three types of experiments in acquired tolerance in the chick embryo are presented, using a technic for embryonic cross transfusion developed in our laboratory: (1) Tolerance to homografts induced by this method is variable in degree and permanence. The method is most effective when applied to chick embryos of twelve to eighteen days' incubation. (2) After embryonic cross transfusion a significantly greater incidence of permanent homograft take is obtained when the test graft is performed between the chicks when they are one day of age rather than when they are two weeks old. (3) When repeat homografts are performed between the same individuals, first at two weeks of age and then at fifteen days of age, the two-week graft invariably sloughs first and there is a significant incidence of primary graft survival in the face of secondary graft slough. This situation holds true in chicks subjected to embryonic cross transfusion as well as in untreated chicks. The possibility that under certain conditions graft adaptation as well as host tolerance may be responsible for homograft survival is suggested. The tentative concept of one of us (J. A. C.) with regard to the total mechanism is presented. The use of homografts in adults from a palliative aspect is discussed.

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DISCUSSION

Emile Holman (San Francisco, Calif.): These fascinating experiments, complicated but meticulously performed by Drs. Cannon, Terasaki and Longmire, carry me back to certain vital experiences thirty-six years ago. A small boy, run over by a motor truck, had suffered the loss of all the skin of one leg from pelvis to ankle. In a burst of youthful enthusiasm—despite the ready availability of the patient's own skin from the trunk and other three extremities—we placed pinch grafts of skin from his mother, whose blood matched his perfectly, on the large granulating surface, placing some 600 grafts in all. They took beautifully and we surveyed our handiwork with overweening pride. That was our second mistake. After about three weeks things began to happen; the grafts lost their beautiful, healthful look and began to melt

away. An annoying eczematous dermatitis with scaling, cracking skin covered the entire body including the scalp. The patient began to bleed from the exposed mucous surfaces and from his rectum. Soon nothing remained of the original skin grafts except the little white islands of subcutaneous tissue submerged in a sea of edematous granulation tissue. Some six weeks later I had an inspiration and removed these little islands of foreign protein with a curet, and within ten days the patient's leg was ready for grafting with his own skin and the widespread dermatitis had completely disappeared. A complete epithelial covering, healthy and permanent, was soon achieved.

This led to a number of experiments in severely burnt patients in which we demonstrated by microscopic studies of the grafts' advancing edge that homografts took, spread but began to melt away about the twenty-fifth day, and by the thirtieth to fortieth day would disappear. We demonstrated also that when skin from one donor was still present as a living graft, a second application of skin from the same donor failed to take and the second skin grafts just floated off the wound in an outpouring of turbid secretions from the raw surface. We concluded that successful homografting of skin was probably a myth, prevented by sensitization of the recipient to the foreign protein of the donor. It is said that blood transfusions are an example of successful homografting of living tissue. This I seriously doubt. It has been shown that the tagged red cells of transfused blood begin to disappear about the seventh day after transfusion and are all gone by the thirtieth day—victims, it would appear, of the sensitization of the host to the foreign protein of the donor. In the meantime, of course, they have served a useful purpose.

Two years ago there appeared from the University of Minnesota a highly provocative article presenting the experiences of Varco et al. in achieving a successful survival of skin transplanted from a donor to an individual whose blood was deficient in gamma globulins. These same individuals had also shown a high lifelong susceptibility to infections and had led a precarious existence due to colds, frequently complicated by pneumonia. It was postulated that the agammaglobulinemia upset the body's resistance to infection by interfering with its complicated immune and antigenic reactions. A similar explanation was applied to the failure of the body to react to the foreign skin graft, permitting it to take and to survive indefinitely.

Think for a moment what this means. In our efforts to render a patient able to accept a foreign living graft we will probably also destroy his continued healthful existence. Ability to accept a graft may occur only if the body's defense mechanism against a foreign protein is destroyed, which, however, may also destroy his immune reactions to deadly bacteria and fatal viruses. Too much interference with the body's intricate immune reactions may in the long run prove more detrimental than helpful. Perhaps the broad vistas envisioned, once the problem of homografting is solved, of possible organ transplantation constitute a mirage of doubtful value. It may be postulated that solution of the problem is incompatible with the survival of the complex chemical and immune reactions developed by man against a hostile environment. One may postulate further that up to the present time successful homografting may be expected only in identical twins, in embryonic tissues and in primitive organisms. We will await with great interest further observations in skin homografting from Dr. Longmire's laboratories, where these important and exciting studies are being conducted with such painstaking care.

ALFRED A. STRAUSS (Palm Springs, Calif.): I am very much interested in the paper by Drs. Cannon, Terasaki and Longmire. The late Dr. Julius Hess, who was head of the Department of Pediatrics at the University of Illinois, in 1920, and I investigated the question of the homograft problem with the idea of seeing what we could do about curing cretins. We took young puppies and transplanted thyroids from one to the other. They disintegrated and did not take. We always put the thyroid transplant into the thyroid itself where its environment was more normal. Then we took a litter of puppies and cross transfused from donor to recipient, and in several instances, vice versa. We also transplanted thyroid in these young puppies of the same litter. We found in about 50 per cent of them a definite take of the transplanted thyroid which was always transplanted into the thyroid tissue.

Later on we took twenty-one puppies of different litters and cross transfused from five to seven times and got takes in about 70 per cent of the transplantations.

I mention this simply because I believe that cross blood transfusions, especially in the same families, have some merit for the success of homograft interpretations.

WILLIAM P. LONGMIRE, JR. (closing): I think it is quite justifiable to present a study of "chicken surgery," as Dr. Cannon has stated, to a group of clinical surgeons, for I believe it is important that we all have some knowledge of the use and limitations of homografts.

Homografts of cornea, blood vessel, bone and cartilage are now quite frequently used.

It is important that we realize there are essentially two types of homografts: (1) the homostatic graft, which includes grafts of blood vessel, bone and cartilage—grafts which merely act as a scaffolding for the invasion of the host tissues; and (2) the homovital graft, which depends on continued survival and reproduction of the transplanted

cells for its value. It is the homovital graft to which Dr. Cannon refers.

Dr. Cannon did not have time to go into detail with respect to the phenomenon of "tolerance." This is one of the most fascinating new "chinks" that has been broken in the "armor" of the homograft problem. Billingham, Medawar and Brent are responsible for collecting evidence from a number of observers and advancing the theory of tolerance. This theory postulates that any antigenic substance which would normally stimulate an immune reaction in adult life will, if injected during the embryonic phase, produce a state of tolerance instead of an antigenic reaction. In other words, skin that is grafted to adult birds or humans will provoke an immune reaction with destruction of the graft, whereas the same tissue transferred during the embryonic stage in birds, and supposedly in humans, could make the recipient tolerant to that particular tissue forever.

This is a fascinating new concept. If an individual is challenged by an antigen at a particular stage in his development, he does not react with an immune reaction but develops permanent tolerance to the particular antigen.

We are somewhat at variance with Billingham and his group, who attempt to explain acceptance of homografted tissue solely on the basis of recipient tolerance, for we believe that the graft itself may vary in its antigenicity and adaptability.

I think Dr. Cannon believes quite strongly that not only does the host play a part in graft acceptance, but also the results and the experience presented here support the view that there is also some change which takes place in all the tissues of the body from embryonic to adult life. This change explains in part the greater success observed in homografts of embryonic or early post-hatching tissue as compared to adult tissues.

Alson R. Kilgore (Belvedere, Calif.): There is one type of homograft which has been known for a great many years and which is successful in a high percentage of cases, and that is the grafting of malignant tissue from one individual to another.

It occurs as an exciting possibility that researches such as that of the authors may help to throw light on the fundamental differences of behavior between malignant and normal tissue.

Jack. A. Cannon (closing): I would like to thank the discussers for their remarks. I was very interested when I recently read Dr. Holman's original paper, because I have not seen or heard of an instance of such allergy to a homograft since that time.

We do think that maybe one of the groups of chickens bearing homografts might not be quite as healthy as those which rejected them. However, we have not yet subjected this impression to statistical analysis.

With reference to Dr. Holman's comments on red cells, it has been my impression that the evidence indicates transfused red cells have about the same life span as autotransfused red cells. I know of no evidence indicating that any reaction occurs in the absence of mismatching. I have explained this absence of a homograft reaction on the basis that homotransfused red cells are anucleated, i.e., the factors responsible for inciting the homograft reaction are in the nucleus of the cells.

I was utterly fascinated by Dr. Strauss' remarks because I think it might turn out that he, as a surgeon, was the first man who induced tolerance.

In response to Dr. Kilgore's remark, I must say that for many years we also have believed this work has important bearing on the cancer problem. Indeed, we have been able to obtain research money on the basis of such justification.

Finally, it is of interest to note that there is a method which permits survival in adult animals. However, along with what Dr. Holman stated, the method involves alterations of the host.

It has been shown that heavily radiated mice can be salvaged by homografts of marrow cells given intravenously. In addition, it has been shown that skin homografts, isogenetic with the marrow homografts, can be applied later to this same host with a high incidence of permanent survival of the skin homograft. Clinical applications of this method can be thought of, but obvious philosophic and ethical objections automatically arise. Consequently, it would seem of great importance to search for a way to modify the tissue to be grafted rather than doing such damage to the future host. Fortunately for my own peace of mind, I am unable to agree with or even appreciate Dr. Holman's profound pessimism with regard to the future of this problem. In fact, his repetition of his original conclusion written in 1924 came as a complete surprise. Most authorities on this subject the world over, as well as surgeons of the eminence of Drs. Blalock and Longmire, believe that the solution of this problem, and hence its clinical application, is within the realm of scientific possibility.

Some consolation for the investigators in this field might also be taken from the words of Dr. Stephan Paget who wrote in 1896, "Surgery of the heart has probably reached the limits set by nature to all surgery: no new method and no new discovery, can overcome the natural difficulties that attend a wound of the heart."

Some of us are even naive enough to believe that the homograft problem may be slightly closer to eventual solution now than the problem of heart surgery was in 1896.

The Management of Visible Hemangiomas

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IN 1949 the Departments of Dermatology, Radiology and Surgery at the University of California Medical Center in San Francisco decided to institute a group project to study the visible hemangioma problem.

We started this project because we were confused. A review of the literature at that time failed to produce a practical classification of these tumors. The histopathology was frequently misinterpreted. Good results were claimed by both those who used homeopathic methods of therapy and those who were radical, as well as those who said no treatment of any kind was necessary. We were also unhappy with our own results.

We met as a group at monthly intervals to examine, diagnose and decide upon the best method of treatment for each case and to evaluate the results of the treatment. Early in the study, all recognized methods of treatment were used. These included CO_2 snow, P32, injection of sclerosing solutions, electrocoagulation, multiple suture, radium and x-ray therapy, and surgical excision. No radical application of these methods was carried out since we believed that essentially all hemangiomas are benign growths.

It was also decided to allow some of the hemangiomas of infants and young children to go without treatment. Patients were observed at intervals of from one to twelve months, depending upon the severity of the lesion and the anxiety of the parents. After observing that most of these lesions involuted spontaneously during the first four years of the study, it was decided to stop all definitive treatment on this group. (Figs. 1A and 1B.)

* Deceased.

The study is far from complete. However, we believe that our evidence of spontaneous involution, along with the convincing results of several other authors, justifies a plea for conservatism in the management of growing hemangiomas in infancy and childhood. Not all authors agree with this view.

As a result of our study of over 700 cases, the following classification appeared more practical than other previous clinical or histologic classifications.

I. Involuting (infantile)
   - A. Superficial (strawberry, capillary)
   - B. Combined: superficial and deep (capillary and cavernous)
   - C. Deep (cavernous)

II. Non-involuting (adult)
   - A. Port-wine stain (nevus flammeus)
     1. Without subsequent growth
     2. With subsequent growth
   - B. True cavernous
     1. Without subsequent growth
     2. With subsequent growth (puberty, pregnancy)
   - C. Venous racemose aneurysm and arteriovenous fistulas
   - D. Diffuse hemangiomatosis
     1. With gigantism
   - E. Spider angioma
   - F. Angiofibroma
   - G. Diffuse telangiectasia
   - H. Malignant (rare)
     1. Hemangioendothelioma
     2. Hemangiopericytoma

In our series more than 200 untreated patients with the involuting type of hemangioma have been followed up. Without exception, spontaneous involution has occurred or is occurring in all these cases. Furthermore, many patients treated earlier by accepted methods

FIG. 1A. Patient P. S. Shows lesion in infant at four months of age.

FIG. 1B. Same patient at three years of age; marked involution, no treatment.

have experienced involution in exactly the same manner as those who have had no treatment, casting doubt upon the effectiveness of some forms of treatment. (Figs. 2 and 3.)

In the smaller lesions no residual evidence of the hemangioma may remain. However, in the larger lesions excess wrinkling and bagginess of the skin can be expected. In these, surgical correction is recommended during the preschool period, at about five years of age. (Fig. 4.)

The non-involuting types require treatment whenever practical. From our observations, most methods of treatment for the port-wine stain (nevus flammeus) give results which only substitute one deformity for another. Radiation of sufficient degree to cause disappearance of the vascular elements will cause both early and late permanent tissue changes. CO_2 snow also damages normal tissue, resulting in surface scarring. Tattooing has not been acceptable from our viewpoint.

Excision and shifting in of adjacent whole thickness skin and subcutaneous tissue gives excellent results in selected cases. However, when normal features such as the eyelids and the angle of the mouth are distorted, the resulting deformity can be more pronounced than the original hemangioma. Application of make-up (i.e., Covermark) is frequently the best solution of the problem.

The port-wine stains that later undergo thickening and nodular growth in adult life require surgical excision. Closure of the defect is carried out by shifting in adjacent tissues or skin grafting.

True cavernous lesions may be quite large and deforming. These lesions may also show later growth at the time of puberty or during pregnancy. Surgical excision is the procedure of choice whenever possible. Injections of sclerosing solution may be of value in selected cases. (Fig. 5.)

Venous racemose aneurysms, arteriovenous fistulas and cases of diffuse hemangiomatosis may present almost insurmountable therapeutic problems. Although few are cured, some benefit can be obtained in selected cases.

Angiofibromas should be excised for the best cosmetic result.

Spider angiomas can usually be eradicated by central vein cauterization.

Diffuse telangectasia is not usually benefited by local therapy. Treatment of associated disorders such as cirrhosis may result in marked improvement.

The rare malignant lesions (none of which have been seen in this series) are treated as are other malignant neoplasms.

SUMMARY

We believe that spontaneous involution of the most common hemangiomas of infancy and childhood must be accepted as a fact. However,

2A 2B

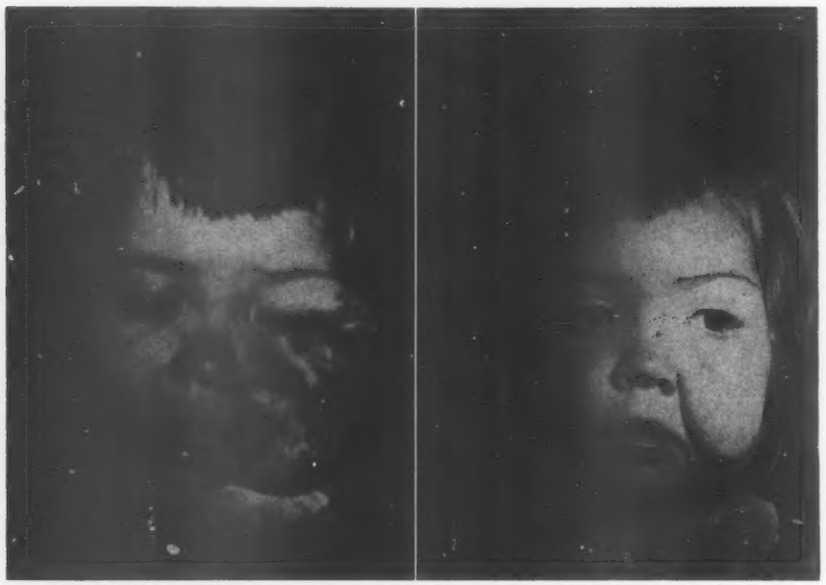
FIG. 2. Patient P. Mc. A, infant at six weeks of age. Five injections with sclerosing solution given from six to eleven months of age with questionable improvement. B, completed involution at the age of six years.

3A 3B

FIG. 3. Patient D. Mc. A, infant at the age of three months with marked ulceration. Treated by radiation with questionable effect. B, final involution and scarring at the age of five years.

in some cases involution progresses slowly, while in a small percentage regression does not occur.

When surgery is forced by parental pressure (Fig. 6) and is attempted during the growing phase of the large involuting type of hemangioma, it can rarely be completely excised and growth will continue in the residual vascular tissue until the latent period is reached. If operation is delayed until involution has started, then one may perform a more conservative excision, as the residual vascular tissue will involute spontaneously. If early therapy is required to placate the parents, some



4A 4B

FIG. 4. Patient M. G. A, large facial lesion at the age of fifteen months. Injection treatment and CO_2 applications attempted without improvement. B, involution present at the age of four years.

5A 5B

FIG. 5. Patient M. M. Treated by radium, sclerosing solution and CO_2 snow in childhood. B, result after many operations. Reconstruction by means of abdominal pedicle flap for forehead and cheek; free full-thickness grafts to eyelids.

homeopathic method of treatment at infrequent intervals should be carried out.

It is also recognized that plastic surgery is necessary to remove the residual cosmetic disfigurements of the large hemangioma in the preschool period. However, surgery which is performed after a number of years following the involution of large hemangiomas is much simpler and less dangerous than when attempted in infancy. Furthermore, we believe

6A 6B

FIG. 6. Patient S. F. A, deep hemangioma after cessation of growth. Parents forced surgery at nine months of age. B, after partial excision and continued involution at the age of four and a half years.

that radical methods of treatment which may damage or prevent the growth of normal tissues are contraindicated.

A discussion of the treatment for the non-involuting types of hemangiomas is presented.

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DISCUSSION

LEONARD DOBSON (San Francisco, Calif.): There are few subjects in medicine in which there are such emphatic differences of opinion as there are in the management of visible hemangiomas. Textbooks and recent articles both present methods of management which are diametrically opposite. Some surgeons, radiologists and dermatologists are as radical in the attack on hemangiomas as on cancer. Others, just as capable and experienced, claim that the great majority of hemangiomas involute spontaneously during the first five years of life and that the end results in the untreated cases are usually better than in those actively treated.

This excellent study is a good example of the value of group study. Such consultation helps to eliminate certain misconceptions and individual prejudices. It provides the authority which is often required to reassure apprehensive parents when active therapy is withheld.

The classification proposed in this article is excellent because it helps in separating those lesions in which spontaneous regression is to be expected from those requiring treatment.

In the Stanford Tumor Clinic it has been recognized in recent years that the great majority of hemangiomas in babies undergo spontaneous involution. The patients have been examined at regular intervals and the progress of the lesions carefully followed with measurements and photographs. In only a very few instances was active therapy (usually radiation) undertaken and this was usually because of parental pressure, rather than continued growth of the hemangioma.

Dr. Alvin Jacobs of the Stanford Department of Pediatrics published an article on this subject in the January, 1957, issue of *California Medicine*. He reviewed the literature, listing many articles, starting in 1900 in which spontaneous regression had occurred in all or practically all strawberry hemangiomas. He personally followed up 105 patients with untreated hemangiomas and found that 97 per cent completely regressed or had involuted to the point that eventual resolution was certain. To determine the incidence of strawberry hemangiomas at different ages, he examined 1,735 consecutive children. Ten per cent of the babies under one year of age had a hemangioma. The incidence decreased rapidly so that in the age group of five to fifteen years the incidence fell to 1.5 per cent.

I wish to congratulate Dr. Blackfield and his fellow workers for this thoughtful and clear presentation of a subject which was previously so confused. The general rules for management of hemangiomas in babies are: (1) observe carefully; (2) let nature take its course; (3) reassure parents; and (4) use plastic surgery when necessary after the maximum involution has occurred.

IAN MACDONALD (Los Angeles, Calif.): I should like to congratulate the group who did this study for their careful work and excellent observations.

Although we have not done any accurate analysis of our series of hemangiomas, Dr. Guiss and I have seen more than 500 infants with this benign neoplasm since 1943, and some are really difficult problems. I know this because I have administered the radium therapy for the Childrens Hospital in Los Angeles over that period of time.

I have realized for some years that the majority of these neoplasms will regress spontaneously. However, there are a small percentage of them in which the non-involuting characteristics are apparent, usually at about the age of ten to eighteen or twenty months.

Bleeding and erosion and infection, in our experience, occasionally cannot be managed by antibiotic ointments or conservative local measures.

I, too, should like to make one emphatic remark: if radiation is given carefully and in small amounts, over a considerable period of time, it can have an effective result, and in the proper amount does not produce any possible damage to normal tissue. Usually that means staying below a tumor dose of 500 r. That, for example, will not produce any disturbance even in such critical areas as epiphyseal tissues.

The objective that we have in mind is to accelerate the natural history of these neoplasms. That is, to do such things, in a dilatory fashion, which will augment the natural involutionizing tendency of these neoplasms; and to treat the emotional status of the parents or, even more difficult at times, of the grandparents.

In 1945 we saw a ten month old child who had had progressive involvement of certain areas since about three months of age. The areas around the lips and chin were repeatedly becoming eroded and infected, sometimes due to trauma, and I will suggest that even though there is a possibility that such widespread involvement might regress, treatment will be effective in hastening that involutionary process. I think that if treatment is properly given there will be fewer residual fibrous areas that require surgical excision when the child is five or six.

This child was treated by a combination of three methods. First, we injected the areas with a sclerosing agent. SyInasol® is the one we happened to prefer. Some of the areas were given small doses of x-ray therapy, none of them receiving more than 300 r. Finally, at about three years of age, .25 mc. radon seeds were implanted in the residual areas, a perfectly simple procedure which is done in the office under local anesthesia.

This girl had a good result at age seven.

Many of these hundreds of children we have seen have been of this variety. They have been sent to us because of the obvious progressive nature of the neoplasms. In only eight or nine have we had to perform eventual surgical excision on residual areas.

In short, my reaction is that although the majority of these lesions do not need treatment, the progressive variety will benefit by careful dilatory treatment.

W. S. Kiskadden (Los Angeles, Calif.): The report by Drs. Blackfield and confreres numbering 700 cases is impressive. I believe that this carefully observed and collected series deserves not only considerable credit and praise but also respect for the observations obtained.

The conclusions that naturally arise after seeing these results is, of course, obvious. This would be to permit all hemangiomas to proceed eventually to involution, feeling perfectly confident that they are going to disappear gradually. The final problem would be simply the excision of redundant or involved skin. However, I cannot believe the problem is as simple as this in the average clinic or surgical group. The reason for such a conclusion is that some three years ago, in conjunction with a number of other Los Angeles plastic surgeons, I heard the preliminary report by Dr. Blackfield on this same subject. We, too, at that time were very much impressed, and brought home our observations with a determination to try them out at the Childrens Hospital Clinic. However, at that institution the dermatologic service, which is not connected with the plastic service, usually saw the cases first and either elected to use injection, carbon dioxide snow or, in occasional cases, some form of radiation or radium therapy. When response to such therapy failed to stop the growth, the cases were referred to the plastic surgical department. Although it was our primary inclination to continue to watch them, we did not have the experience or the background of cases which had involuted to show the parents and, therefore, with the pressure upon our group exerted both by the dermatologic department, the family and the relatives and friends, as Dr. Macdonald has previously suggested, coupled with our own unsureness, we often proceeded with surgery. We believe our surgical results have been entirely satisfactory, and it is quite possible that the trauma of the removal has expedited more quickly than mere time the subsequent involution.

I cannot be certain in my own mind that the fact of eventual involution of these tumors when extended to pediatricians, surgeons and medical men, generally, may not be a dangerous philosophy. It has not been at all unusual for us to have patients who have been told that disappearance would undoubtedly occur, and in whom, under indifferent management, ulceration, bleeding, subsequent scarring and, in two cases, death has occurred. It is only fair to point out, however, that these cases occurred before the advent of antibiotics, and treatment at that time was not adequate.

I believe the following case illustrates the problem I have just pointed out. A pediatrician told the patient's family not to be concerned; the tumor would involute. The mother, who was caring for her first child, was only too ready to accept this solution. The infant had a rather large hemangioma involving the entire arm, shoulder and a portion of the chest. This condition occurred between his birth and ten weeks of age. At this point, because of the enlargement, excoriation and ulceration, the mother consulted with a roentgenologist in Los Angeles who in turn referred the case to me for treatment. This was an inoperable lesion, and the only treatment that we believed would be wise at

this time was the implantation of radon seeds by the roentgenologist. I have no immediate record of the number of seeds that were implanted, but there was regression four years after the implantation. I have no idea what might have happened to this youngster had the seeds not been implanted. However, because of the ulceration that was present at the time I first saw the patient, I would be of the opinion that waiting for involution to occur would have certainly been a dangerous procedure to employ. Up to the present there is no difference in the growth of this arm, although the radiation therapy may have some effect at a later date.

In 1939 Dr. Wilbur Bailey and myself collaborated on a paper which at this point I would like to disclaim, although at the time it seemed that because of the neglect of these tumors either the implantation of radon seeds, the use of radon packs or radium plaques might be the method of choice.

Today, I believe the opinion about radium and x-ray is definitely on the wane, and it should be used only in specific and definite cases when no other therapy is justified. Two years ago I had occasion to go to Hiroshima to investigate the after-results of the atomic bomb explosion. The investigative work which has been brought forth since that time, in cooperation with reports from other areas, seemed to indicate that the use of x-ray and radium, even in rather limited to small dosages, may be a dangerous therapeutic agent, particularly from a standpoint of genetics.

Some of you may have had occasion to read recently of the muskrats that had been feeding on the vegetation along one of the tributaries of the dump areas of the Atomic Energy Commission's institution in Tennessee. On autopsy all of them were found to have advanced carcinoma and sarcoma, apparently derived from the waste products absorbed by the plants along the river bed.

In conclusion, I think it is important to point out that the method produced by the authors of this paper has proved unquestionably the value of watchful waiting with the probability of involution. I do not believe that it is their intent to suggest that surgery should not be performed whenever it is feasible, and on this basis certainly the method presented is the one that should be the procedure of choice.

DR. MEYER (New York, N. Y.): We learned in the Tumor Clinic at New York University and at the postgraduate school that it is definitely most important to treat the parents, as has been expressed in the authors' paper. We have found that by showing photographs taken every few months to the parents (especially colored photographs) we were able to control the families.

Occasionally it is necessary to use placebo treatment. Frequently we have the mother bring some dry ice into the clinic and apply it just long enough to have the baby cry and satisfy the mother. It does not cause any scarring.

In a big city like New York it has been our experience that these parents and grandparents will go from clinic to clinic until they find a doctor who will do something. They are not satisfied to just sit still.

When further treatment becomes necessary, after waiting for the important resolution and involution, there is no routine treatment for these lesions and we have to individualize every single case and select the method or combination of methods of treatment that is important.

We learned many years ago that if there is evidence that involution will not take place, as Dr. Macdonald stated, and if surgery does become necessary, as Dr. Kiskadden has said, the injection of 1,000:10,000 adrenalin® solution subcutaneously around these lesions is a great help.

I would like to warn against one thing. In some large clinics and hospitals they have instituted special clinics which they call Injection Clinics. The members of those clinics often become overly enthusiastic, and if so, harm can be done by the use of injection in cases in which it is not indicated.

HARRY M. BLACKFIELD (closing): I appreciate the dilemma of any physician who has to face the problem of placating the parents of a child with a hemangioma.

I would like to discuss one or two things. It has been our belief, and in this our radiation consultant agrees, that if radiation of sufficient intensity is given to destroy adult vessels in a hemangioma, it will also destroy normal tissues. During the past two years the x-ray department at our institution has stopped treating hemangiomas.

I appreciated the report of Dr. Dobson on his separate study which has also been going on for a number of years. It was also of interest to hear that Dr. Meyer and his group in New York were conservative in their treatment of hemangiomas.

Problems in Diagnosis Associated with Obstructive Neonatal Jaundice

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From Childrens Hospital, Los Angeles, California.

IN 1935 Ladd [1], in discussing the differential diagnosis of prolonged neonatal jaundice, stated that after four to six weeks of age consideration could be limited to the following major conditions: biliary atresia, erythroblastosis fetalis, hemolytic sepsis and syphilis.

At the present time the problems in differential diagnosis presented by the icteric infant are different, although not less exacting. Hemolytic disease of the newborn has become a field of conditions usually readily identified by the Coombs' phenomenon in the infant and the studies of Rh and ABO factors in the parents. Sepsis is less prevalent and the therapy affects the clinical picture more readily, making the diagnosis less difficult. Neonatal syphilis is rarely seen today. New disease processes now included in the differential diagnosis of obstructive neonatal jaundice include galactosemia and cytomegalic inclusion body disease. Both are uncommon and in both a specific diagnostic test is available to aid in early recognition of the disease.

The present differential diagnostic possibilities in cases of apparently obstructive neonatal jaundice, when the surgeon first examines these infants, are ordinarily confined to three conditions. These include the well established anomalies of extrahepatic and intrahepatic atresia and a group of conditions producing a clinical syndrome referred to as "neonatal hepatitis." The present study is directed toward a comparison of the methods of diagnosis in these cases, particularly in regard to the preoperative differentiation between biliary atresia and cases of the neonatal hepatitis syndrome. The ultimate diagnostic tests to differentiate extrahepatic biliary atresia from intrahepatic biliary atresia remain operative in nature.

The critical period when the distinction between biliary atresia and the neonatal hepatitis syndrome must be made lies between the fourth and eighth weeks of life. Attention has been directed in all instances to the history, physical findings and laboratory data at this specific period, rather than to data obtained later in the course of either disease process.

CLINICAL SERIES

The present study includes an analysis of fifty-four* cases of extrahepatic biliary atresia, eight cases of intrahepatic biliary atresia and twenty-three cases showing the neonatal-hepatitis syndrome. These infants were admitted to Los Angeles Childrens Hospital during the twenty-year period, 1937 through 1956. The cases from the initial decade are largely from the autopsy files due to strict adherence to the criterion to be outlined shortly, while those in the second ten-year period come from both autopsy and clinical material. This may distort the relative incidence of these diseases, but does not alter the diagnostic aspects which are the subject of the present study.

Extrahepatic biliary atresia was demonstrated at operation or autopsy in all cases. Intrahepatic atresia was believed to be only well established by autopsy or multiple biopsy, and all the cases included in the present series had either a complete absence of biliary epithelium (four cases) or a major intrahepatic block (four cases).

Cases in the neonatal hepatitis group were selected on the basis of clinical data, operative observation, roentgenographic and biopsy studies and autopsy material. In this group have been included infants with protracted jaundice beginning prior to the fourth week of life who

* Including two cases of extrahepatic biliary stenosis.

gave evidence of hepatic disease of an obstructive type through liver function tests, stool and urine examinations, and in whom patency of the extrahepatic tree was demonstrated by surgery, cholangiogram or clinical recovery. No evidence of hemolysis, sepsis, toxic exposure or local infection was present. In some of these cases a tentative diagnosis of viral hepatitis was made. A gross inspissated bile "plug" was not visualized in any of the patients in this group at surgery or autopsy. The liver biopsy findings in this group were not uniform. Neonatal hepatitis should be regarded as a useful clinical syndrome, not as a single pathologic disease process.

TABLE I
NEONATAL JAUNDICE: STATISTICAL DATA

Data	Biliary Atresia (No. of Cases)		Neonatal Hepatitis (No. of Cases)
	Extra-hepatic	Intra-hepatic	
Sex:			
Male	23	2	18
Female	31	6	5
Race:			
White	52	8	19
Negro	2	0	4
Mexican-American	3	0	4
Premature infants	9	1	6
Mongoloid infants	0	0	3

Among the eighty-five infants were twenty-three males and thirty-one females with extrahepatic biliary atresia, two males and six females with intrahepatic biliary atresia and eighteen males and five females with neonatal hepatitis. Birth weights were less than 2,500 gm. in fifteen infants in the group. In the neonatal hepatitis series three infants showed definite mongoloid features and a fourth had mental retardation of unknown etiology. The racial background, environmental factors, incidence of prematurity and incidence of mongoloid features in these groups are outlined in Table I.

HISTORY

Among the fifty-eight infants with biliary atresia for whom an early history could be obtained, thirty-five (65 per cent) were described as continuously jaundiced from birth. The remaining twenty-three were first noted to be jaundiced, in approximately equal numbers, on each successive week during the first month. (Table II.) In one infant with associated cyanotic congenital heart disease, jaundice was not noted during life. In the neonatal hepatitis group five patients (22 per cent) were jaundiced from birth, and the remainder became icteric during the subsequent four weeks. (Table II.) Infants with jaundice appearing for the first time after the fourth week were not included in the series.

TABLE II
ONSET OF JAUNDICE

Time of Onset	Biliary Atresia (No. of Cases)	Hepatitis (No. of Cases)
From birth	35 (60%)	5 (22%)
First week	7 (12%)	2 (9%)
Second week	7 (12%)	5 (22%)
Third week	2 (4%)	4 (18%)
Fourth week	7* (12%)	7 (30%)

* No jaundice noted in one.

Stools were acholic, white or grey, continuously from birth in forty-six (90 per cent) infants with biliary atresia for whom an apparently accurate history could be obtained in this respect. The remaining five infants had pigmented stools described at one or more periods during the initial eight weeks of life.

The feces in the neonatal hepatitis group were noted to be completely acholic from birth in eight infants (35 per cent), initially acholic and later pigmented (in at least one stool) in seven cases (30 per cent), and continuously slightly pigmented in eight cases (35 per cent). All observations in this regard were confined to the initial two months of life.

Infants in both biliary atresia and neonatal hepatitis groups were described as passing dark urine from birth. Additional symptoms in both groups were few. Four patients in each series were febrile prior to entry or soon thereafter. Two patients with biliary atresia had abnormal bleeding, one from the gastrointestinal tract and one from the umbilical vessels. Other symptoms noted were related to associated congenital abnormalities or prematurity rather than to primary diseases of the hepatic or biliary systems.

PHYSICAL FINDINGS

All infants studied were intensely jaundiced. An enlarged liver was present in all cases of extrahepatic atresia and neonatal hepatitis. In one-half of the patients in each series the liver was palpated 4 cm. or more below the rib margin. In the cases of intrahepatic atresia, however, the liver was not palpable in one infant and was less than 3 cm. below the costal margin in the remaining cases. These findings were again confined to the cases examined between the fourth and eighth week of life.

TABLE III
HEPATIC FUNCTION STUDIES

Studies	Biliary Atresia (No. of Cases)				Neonatal Hepatitis (No. of Cases)	
	Up to 2 mo.		Past 2 mo.			
	Positive	Negative	Positive	Negative	Positive	Negative
Cephalin flocculation	2	12	8	5	4	8
Thymol turbidity	1	4	5	7	2	7
Cholesterol	2	1	3	0	2	0
Alkaline phosphatase	3	4	8	3	2	3

Splenomegaly was not apparent to examination in thirty-four of the forty-four infants with biliary atresia examined at this age. Among the ten infants with palpable spleens none were described as extending more than 2 cm. below the costal margin. Among the twenty-one patients with neonatal hepatitis seen at the same age, eleven had no evidence of splenomegaly, but in the remaining infants the enlargement was marked. In four of these the spleen extended more than 4 cm. below the costal margin.

Neither group had cutaneous telangiectasis, dilated abdominal veins, lymphadenopathy or ascites during this age period. Later in the course of untreated biliary atresia, or neonatal hepatitis progressing to cirrhosis, these signs became apparent.

ROUTINE LABORATORY STUDIES

Significant anemia was not present between four and eight weeks, the critical period of differential diagnosis, in either the biliary atresia or the neonatal hepatitis groups. Varying degrees of transient leukocytosis were encountered in both series. Routine urinalyses were unremarkable.

The majority of tests for hepatocellular liver damage became positive in the untreated cases of biliary atresia at approximately four months of age. Results of the routine tests of hepatic function performed in cases of biliary atresia during the critical period, biliary atresia later in the course of the disease and the neonatal hepatitis group are found in Table III.

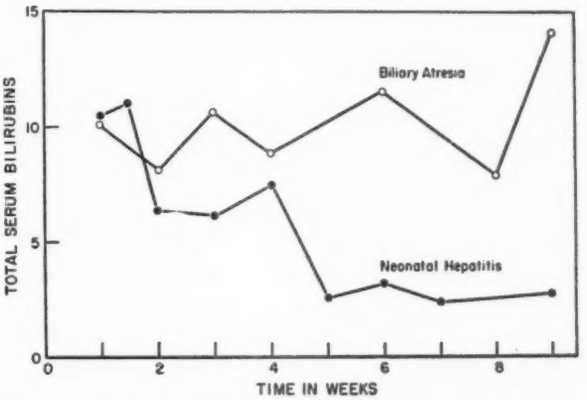

FIG. 1. Average of weekly serial bilirubins. Total serum bilirubin levels in infants admitted between four and eight weeks of age. The initial determination in each group is that performed at the time of admission, and subsequent determinations at weekly intervals thereafter.

Direct and total serum bilirubin determinations were performed on eighty-three infants. In twenty-four, weekly serial studies were carried out, and averages of the determinations in this group were used to plot the total serum bilirubin curve for infants with biliary atresia and for those with neonatal hepatitis syndrome. (Fig. 1.) The individual curves for these infants in each series are shown in Figures 2 and 3.

The bilirubin and urobilinogen content of the urine and feces was examined during the critical diagnostic period in seventy of these infants and the results are shown in Table IV. Bile pigments were completely absent from the feces in forty-four of the forty-eight infants with biliary atresia, and in fourteen of the twenty-two infants with neonatal hepatitis. Urinary bilirubin levels were elevated in all cases of atresia as well as in fourteen of the fifteen cases of neonatal hepatitis. The unusual laboratory findings were generally found among infants in the eighth week of life when serum and tissue bilirubin levels were markedly elevated.

ROENTGENOGRAPHIC STUDIES

Operative cholangiography was performed in twelve patients in this series. (Table v.) In many other infants attempts to inject the biliary system were completely unsuccessful.

In six cases of biliary atresia some elements of the biliary tract were successfully demonstrated by injection technic. This included two patients with intrahepatic atresia. In five infants with neonatal hepatitis the entire biliary tract was outlined by cholangiography at surgery. All such attempts at visualization in the neonatal hepatitis group were successful when attempted. A solution of 70 per cent diodrast® was employed as the contrast medium, and the injection performed through the gallbladder.

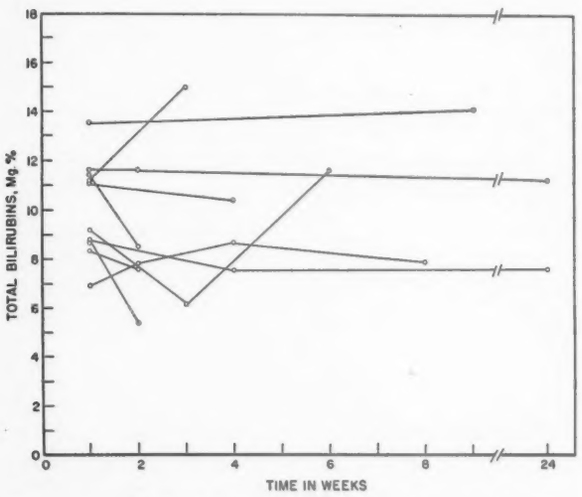

FIG. 2. Individual infant's serial weekly total serum bilirubin curves in patients with biliary atresia. The initial determination in each case is that performed at the time of admission, some time between the fourth and eighth week of life.

TABLE IV
PIGMENT STUDIES: FECES AND URINE

Studies	Biliary Atresia (No. of Cases)	Neonatal Hepatitis (No. of Cases)
Feces		
Bile pigment:		
Present.....	4	8
Absent.....	44	14
Urine		
Bilirubin:		
Positive....	29	14
Negative...	0	1
Urobilinogen:		
Present.....	4	2
Absent.....	23	9

Intravenous 20 per cent sodium iodipamide, or cholografin,® was administered to sixteen patients in an attempt to delineate the biliary tree. Among fifteen of these patients there was no visible opacification in the hepatic region, despite the use of dosages as high as 1.6 cc. per kg. of body weight, five times the usual adult dosage. This group included twelve cases of biliary atresia and three infants with the neonatal hepatitis syndrome. A fourth case of neonatal hepatitis, in which the serum bilirubin level was already falling, showed delayed filling of the gallbladder.

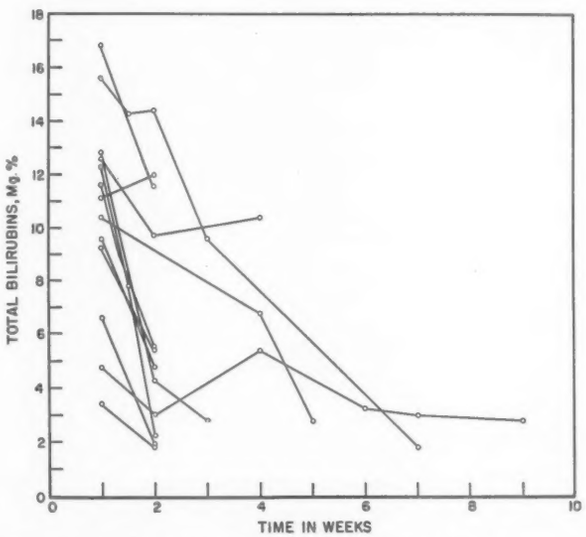

FIG. 3. Individual infant's serial weekly total serum bilirubin curves in patients with neonatal hepatitis. The initial determination in each case is that performed at the time of admission, some time between the fourth and eighth week of life.

SPECIAL DIAGNOSTIC STUDIES

Procedures employed in an attempt to differentiate biliary atresia from the neonatal hepatitis group during the initial eight weeks of life include the administration of cholagogues and duodenal drainage with or without cholagogue administration. The diagnostic procedures employed during the initial phase of abdominal exploration included liver biopsy and hepatic exploration with multiple needle aspirations.

Oral choloretic solutions consisting of 25 per cent magnesium sulfate or intravenous cholo-

retic substances such as decholin® were administered daily to six infants during the critical diagnostic period. It was believed that this stimulation might reduce the cholemia or produce fecal pigment for analysis in the neonatal hepatitis cases. Neither of these results were obtained. Ultimately three of these infants were shown to have biliary atresia and three were included in the neonatal hepatitis group.

TABLE V
ROENTGENOGRAPHIC STUDIES

Studies	Biliary Atresia (No. of Cases)	Neonatal Hepatitis (No. of Cases)
Intravenous cholografin:		
Visualization	0	1
Non-visualization	12	3
Operative cholangiogram:		
Visualization	6	5
Non-visualization	1	0

Duodenal drainage was carried out eight times on infants with neonatal jaundice. Four of these intubations produced fluid containing at least a trace of bilirubin. Three of these infants subsequently were shown to have neonatal hepatitis, and the fourth had extrahepatic biliary hepatitis. Among four patients in whom duodenal drainage was negative for bile pigment, all ultimately were proved to have atresia. The addition of magnesium sulfate through the duodenal drainage tube produced intestinal fluid which was negative for pigments, even in the cases positive prior to its administration.

Liver biopsy was performed in forty-five instances on forty-three patients in this series. Rapid sections during the operative procedures were helpful in determining the presence or absence of biliary ducts. They were not of aid in distinguishing between extrahepatic atresia and neonatal hepatitis.

In three infants, following the usual exploration of the porta hepatis or as a separate procedure, the medial inferior surface of the liver was aspirated to a depth of 5 cm. at multiple points with a No. 17 gauge needle. No bile was encountered and no chamber located by irrigation which could be injected for roentgenographic study.

ERRORS IN DIAGNOSIS

Eight cases of biliary atresia were not surgically explored. (Table VI.) In six instances this was due to a definite error in diagnosis. Three of these infants were presumed to have "hepatitis," and one a partial obstruction apparently due to inspissated bile. The fifth infant had both congenital syphilis and biliary atresia, the latter remaining unrecognized until postmortem examination. Death was due to pneumonia. These patients were seen between 1939 and 1950. The sixth infant in whom atresia was unrecognized clinically had severe cyanosis secondary to multiple cardiac septal defects. Autopsy revealed biliary atresia in all cases.

TABLE VI
OBSTRUCTIVE NEONATAL JAUNDICE: ERRORS IN DIAGNOSIS
(1937–1956, EIGHTY-FIVE CASES)

Data	Biliary Atresia (No. of Cases)		Biliary Stenosis (No. of Cases)	Neonatal Hepatitis (No. of Cases)
	Extrahepatic	Intrahepatic		
Total number of cases	52	8	2	23
Surgical exploration	44	5	2	8
No surgical exploration	8	3	0	15
Errors in diagnosis	6	3	0	8

In the remaining two infants surgery was delayed for approximately one month because of severe diarrhea and duodenal ulcer which ultimately perforated. Death resulted from complications of the other major disease process.

Three of the eight infants with intrahepatic biliary atresia did not undergo surgical exploration. Two were regarded as cases of hepatitis and later of cirrhosis, until the time of autopsy. Exploration was performed in the third infant because of duodenal atresia on the third day of life. Biliary atresia was not recognized at surgery, but established at autopsy following postoperative death. These must be regarded as errors in diagnosis although no effect on the outcome of the case is implied.

Eight of the twenty-three infants with neonatal hepatitis underwent exploration with a preoperative diagnosis of biliary atresia. In five, cholangiogram and liver biopsy were performed without further exploration. In all cases the extrahepatic biliary tree was ultimately demonstrated to be functional. In a ninth infant exploration, liver biopsy and pyloromyot-

omy for hypertrophic pyloric stenosis were performed.

It is not the purpose of this study to analyze operative technics or results of therapy. The two long-term survivals in the biliary atresia series (Table VII) have been previously reported by Greaney, Snyder and Chaffin [2]. Their status is unchanged.

TABLE VII
THERAPY AND RESULTS IN SIXTY-THREE CASES OF EXTRA- AND INTRAHEPATIC BILIARY ATRESIA

Present Status	No Surgical Exploration (No. of Cases)	Exploration Only (No. of Cases)	Exploration and Anastomosis (No. of Cases)
Total cases	11	40	11
Postoperative deaths	0	2	1
Known to be alive:			
At 6 mo	0	30	6
At 1 yr	0	22	4
At 2 yr	0	7	4
At 3 yr	0	3	3
At 4 yr	0	2	3
At 5 yr	0	1	2

Among the eight patients with neonatal hepatitis in whom exploration was performed, there was one postoperative death secondary to hepatic insufficiency. (Table VIII.) Five of these infants have shown definite evidence of cirrhosis since surgery. One of them succumbed to a complication of cirrhosis with necropsy confirmation of the diagnosis of posthepatitic cirrhosis.

Among the fifteen infants in the neonatal hepatitis group who did not undergo surgical exploration, hepatomegaly has subsequently developed in one.

COMMENTS

Among all types of neonatal jaundice, including those secondary to hemolytic sepsis, metabolic disease, hepatic prematurity, local infection, etc., those apparently obstructive in character comprise approximately 5 per cent.

Three-quarters of this relatively small group have some form of biliary atresia, extrahepatic or intrahepatic, partial or complete. The remaining patients defy identification by specific tests. They are jaundiced from birth or shortly thereafter. The icterus is of an obstructive type to all routine examinations. They present no evidence of toxic exposure. Viral hepatitis may be postulated from the history only in a minority. Recovery within six to eight weeks is usual and the incidence of cirrhosis following the acute process is high.

TABLE VIII
MANAGEMENT AND RESULTS IN TWENTY-THREE CASES OF NEONATAL HEPATITIS*

Therapy	Total No. of Cases	Deaths, Type (No. of Cases)			Alive (No. of Cases)	
		Post operative	Hepatitis	Cirrhosis	No Cirrhosis	Cirrhosis
Surgical exploration	8	1	0	1	2	4
No surgical exploration	15	0	0	0	14	1

* Biliary atresia was diagnosed initially in twenty-one patients.

No gross intraluminal "bile plug" has been demonstrated at surgery in this group, in the present series. Microscopic "bile plugs" are no more prevalent in the liver sections of these patients than in sections from patients with demonstrated biliary atresia. As Harris et al. [3] point out, the term "inspissated bile plug syndrome"* suggests an unproved etiology in these cases. The descriptive term "neonatal hepatitis syndrome" seems preferable until etiologic factors are known.

Among the infants in the neonatal hepatitis group there was an abnormally high incidence of males, of prematurity, of Negro racial origin and of Mexican-American parentage. Several authors [4] express the opinion that biliary atresia is a disease of the Caucasian race and "inspissated bile" a disease of the Negro race. Gerrish and Cole [4] state there has never been a report of a "single proven case of biliary atresia in a negro infant." Two cases of extrahepatic biliary atresia in Negroes were present in this series and one of them was confirmed at autopsy. Home environment, economical status and general debilitation may be more important factors than racial origin in the inclusion of infants in the neonatal hepatitis group.

The classic concept of the clinical history of

* This term is more correctly applied to the secondary obstruction of the biliary passages in known hemolytic diseases.

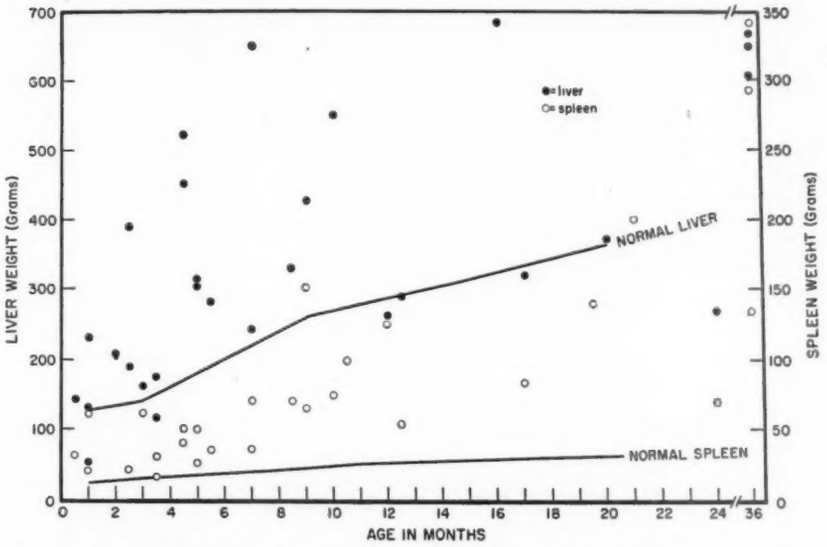

FIG. 4. Gross liver and spleen weights at autopsy on patients with untreated biliary atresia. Massive spleen enlargement was not found prior to the sixth month of life. Hepatic enlargement was variable, but generally progressive with advancing age.

biliary atresia as a progressively increasing cholemia, beginning at birth, was seen in the majority of cases of both atresia and the neonatal hepatitis syndrome. The historical data obtained from the parents was generally of little aid in differentiating between these two groups. Jaundice was not recognized for several weeks following birth in many instances in both groups.

The passage of acholic stools from the time of birth was described in almost all cases of atresia and in many cases in the neonatal hepatitis group. The other significant pattern among the histories of infants with the neonatal hepatitis syndrome was a recorded passage of pigmented stools during the first week of life, with acholic stools thereafter, until the age of four to eight weeks.

Significant and usually progressive hepatomegaly was noted in both the extrahepatic atresia and neonatal hepatitis groups on admission and during the observation period. In the small series of cases of intrahepatic biliary atresia, hepatomegaly was absent or minimal, an interesting factor. A decrease in liver size at any stage of the observation period before two months of age strongly suggested the diagnosis of the neonatal hepatitis syndrome.

Splenomegaly was not present in the great majority of atresia cases, either extrahepatic or intrahepatic, during the critical observation period. It was likewise absent in approximately one-half of the cases of neonatal hepatitis. The remaining infants in the neonatal hepatitis group however had significantly enlarged spleens, some of them massively enlarged. Spleens of this size were not encountered clinically among patients with biliary atresia until four to six months of age when secondary factors such as portal hypertension probably begin to influence the spleen.

Spleen weight in patients with biliary atresia was also studied in the thirty-four cases that came to autopsy. (Fig. 4.) Massive splenomegaly made its appearance only in infants dying after the third month of life. Thus, marked splenomegaly prior to two months of age strongly suggests the diagnosis of neonatal hepatitis syndrome.

Little diagnostic aid was obtained from the routine liver function studies. Abnormalities in serum protein, prothrombin time, cephalin flocculation, thymol turbidity, cholesterol esters, etc., were almost universally absent in both the atresia and neonatal hepatitis groups prior to the third month. Elevated single serum bilirubin levels with a predominant direct component and high alkaline phosphatase levels were found in both groups.

Serial total serum bilirubin levels were more helpful. This has been emphasized by Hsia and Gellis [5] and confirmed by Kiesewetter et al. [6] as well as others. In our series only three infants in the neonatal hepatitis group did not

conform to the general pattern of descending curve of weekly serial bilirubin levels during the observation period; and only four cases of atresia did not produce bilirubin curves which rose or remained stationary during the same period. The confusing descending curves among the four cases of atresia in which this occurred were from very high to moderately high serum bilirubin levels, all above the average range of the neonatal hepatitis group. (Fig. 3.) This is a significant improvement over the routine liver profile studies.

It should be noted that alterations in all of the tests of hepatocellular liver disease become positive in cases of biliary atresia beyond the fourth month and often positive in cases of hepatitis in which evidence of cirrhosis develops.

Duodenal intubation may be of value in efforts to obtain pigmented intestinal content and establish patency of the biliary tract. The use of cholagogues, either oral or intravenous, has not been of aid in this regard.

The "frozen section" examination of hepatic tissue at the operating table has not been helpful in distinguishing between neonatal hepatitis and extrahepatic biliary atresia. On the other hand, it has been diagnostic in cases of intrahepatic biliary atresia with complete absence of biliary ductal epithelium.

Operative cholangiography is the greatest single aid in enabling the surgeon to limit his operative procedure once it is started. All patients in the neonatal hepatitis group had an accessible gallbladder and no attempt to use this for contrast visualization of the biliary tree was unsuccessful. When cholangiography is possible in cases of atresia, it may help in planning the anastomosis.

The value of intravenous cholecystography as a diagnostic study is limited by impaired liver function during the active phase of hepatitis. Visualization of any portion of the biliary system is definite evidence that atresia does not exist, and this may be possible at some time during the course of the disease in patients in the neonatal hepatitis group.

There were seventeen errors of diagnosis in this series, or 20 per cent of the eighty-five cases of neonatal jaundice. (Table VI.) It is of interest that the mistake of withholding surgery from patients with extrahepatic biliary atresia was prevalent in the initial decade of the study. On the other hand, diagnostic error resulting in exploration of patients with neonatal hepatitis have been confined entirely to the second decade of the study.

The diagnoses of intrahepatic biliary atresia and extrahepatic biliary stenosis must be made at surgery. These conditions are clinically undistinguishable from extrahepatic biliary atresia and should be subjected to surgical exploration. It is recognized that in intrahepatic atresia of the type described by Ahrens [7] with no biliary ducts, no anastomotic procedure is feasible. In the other variety, with major intrahepatic duct occlusion, hepatojejunostomy, as performed by Longmire [8], offers the only chance of improvement, although a successful case has yet to be reported. Dissection in the area of the liver hilum is indicated if needle aspiration in this region demonstrates a bile-filled cavity, which can be injected with radiopaque media. In three cases of this series the liver was aspirated to depths of five cm. throughout the inferior surface of the liver without locating such a cavity.

It is important to differentiate successfully between biliary atresia and neonatal hepatitis between the fourth and eighth weeks of life. Diagnosis is hazardous before the fourth week, and surgical exploration performed prior to that time is apparently not necessary for a cure in biliary atresia. After the eighth week the incidence of irreversible cirrhosis in cases of biliary atresia is high, despite successful anastomosis. Unnecessary surgical exploration of patients with neonatal hepatitis may have a deleterious effect on the damaged liver, resulting in a higher incidence of cirrhosis and an increased mortality rate. The clinical differentiation between biliary atresia and the obstructive phase of neonatal hepatitis becomes an effort to find a few isolated indications of the patency of the biliary tract, or of a primary inflammatory liver process in the maze of identical findings and laboratory results.

A rather frequent and extremely difficult diagnostic situation is created when a complete but minute extrahepatic biliary tree is encountered during surgery. Bile is very often absent from the tiny radicles. Is such a case one of hepatitis in the obstructive phase, or is it a problem of intrahepatic atresia with atrophy of disuse affecting the extrahepatic system? Liver biopsy with rapid section in this group may reveal the complete absence of all ductal epithelium; and if this is true in several areas,

the diagnosis of generalized intrahepatic atresia is the most tenable. An operative cholangiogram may be possible in these cases; and if the major intrahepatic as well as the extrahepatic ductal system can be visualized, the diagnosis then lies between hepatitis and intrahepatic atresia involving only the finer hepatic radicles. This should end the operative procedure. In general, a conservative approach to this type of problem seems indicated by the isolated reports [9] (as well as the one case in this series) of late but complete recovery following such operative findings.

A somewhat different situation has been encountered by Longmire, in which the extrahepatic ductal system is minute but has a lumen. This seems occluded, however, in the distal portion of the choledochus. Drainage of the lumen or anastomosis to the duodenum seems indicated in such cases. Nothing has been lost by such a procedure, and in at least one instance such a structure has ultimately enlarged and become an adequate common duct.

The general aim should be to arrive at a diagnosis by the eighth week of life or earlier if possible, and operate at this time. In the few cases in which preoperative error is unavoidable, the operative procedure may be limited in time and trauma to the infant lessened by the use of immediate diagnostic procedures at the operating table.

SUMMARY

Diagnostic possibilities considered in evaluating prolonged obstructive neonatal jaundice have been clarified in the past decade to the extent that the field of diagnostic uncertainty is usually narrowed to include only three conditions, extrahepatic biliary atresia, intrahepatic biliary atresia and the clinical syndrome of neonatal hepatitis.

Surgery may be curative in extrahepatic atresia, is not helpful in intrahepatic atresia and is contraindicated in the neonatal hepatitis group.

Eighty-five patients presenting this diagnostic problem, admitted to the Los Angeles Childrens Hospital during the twenty-year period between 1936 and 1956, have been analyzed. Methods of preoperative and operative differentiations of these conditions are compared. The serial weekly total bilirubin curve, duodenal drainage and the pigment analysis of urine and feces have shown the most accuracy in this regard. Routine liver function studies, intravenous cholecystography, multiple liver aspirations and rapid section liver biopsy have not been helpful. Operative cholangiograms have been the greatest single aid in limiting the extent of the surgical procedure.

Although the exploration of non-surgical cases cannot be avoided completely, it is believed that judicious employment of available technics may at times eliminate an operative procedure, and at other times reduce its length and extent without loss of diagnostic accuracy.

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DISCUSSION

Edwin G. Clausen (Oakland, Calif.): Drs. Norris and Hays have presented the differential diagnosis of neonatal jaundice comprehensively. Our experience at the Children's Hospital of the East Bay in Oakland, although less extensive, has paralleled theirs very closely. A differential diagnosis of prolonged jaundice is essentially between obstructive jaundice due to atresia or stenosis of the bile ducts and neonatal hepatitis. All other causes of jaundice can be ruled out if the proper diagnostic steps are taken. However, as they have shown, neonatal hepatitis and atresia of the ducts are frequently indistinguishable clinically since both will present findings of complete obstruction, and liver function tests in this neonatal period have proved to be undependable. A desire by surgeons to operate upon these infants early before liver damage occurs has met moderate resistance from pediatricians. This is not surprising when one analyzes the number of patients salvaged. A high

degree of satisfaction is lacking even in those few cases of atresia of the extrahepatic ducts which are amenable to surgery. We are interested in knowing how much damage is done when we operate upon a patient with hepatitis. The Boston Children's Center reported forty-two patients with neonatal hepatitis, eight of whom had been operated upon. The only mortality and morbidity was in seven of the eight operative cases. In the Los Angeles group, likewise, the only mortality and morbidity, with one exception, exists in those patients who were operated upon. These patients presumably are completely obstructed, and hence a poor prognosis might be expected.

Pathologists are confident that on permanent sections they can tell us whether the jaundice is due to hepatocellular or ductal obstruction. Clatworthy has recommended diagnostic laparotomy in obstructive jaundice in infants. Exploration is performed under local or cyclopropane anesthesia. If a gallbladder is present, a cholangiogram is taken; otherwise, a liver biopsy only is taken. In this manner no damage will be done to minute, non-functioning but intact, extrahepatic ducts. If the microscopic sections reveal the presence of extrahepatic obstruction, the infant is returned to surgery for an attempt at more definitive surgery.

I would like to ask the authors if they have had any experience with cortisone as a cholagogue in neonatal hepatitis. There have been cases in which the steroid produced a marked drop in the serum bilirubin and rather precipitously relieved the obstruction, and hence could be used as a therapeutic test.

WILLIAM J. NORRIS (closing): I would like to say just one word before asking Dr. Hays to close the discussion, and that is that the tendency to explore these children early, I believe, will continue because, in my humble opinion, the cause of morbidity is not the exploratory laparotomy.

I believe that if you are going to open the abdomen enough to perform cholangiography and take a biopsy of the liver, you can just as well insert your finger down to the porta hepatis and do quite a thorough exploration, without increasing the trauma to the child sufficiently to make any difference.

DANIEL M. HAYS (closing): It is true that we have operated upon these children progressively earlier. Prior to four weeks of age, however, I think the diagnosis is extremely hazardous. Apparently the liver changes are not far enough advanced so as to make a cure improbable when anastomosis is possible. Therefore, I do not think we will operate any earlier than four weeks. Actually, irreversible changes have not been reported prior to eight weeks, so probably we are safe in waiting that long.

The publication of the Boston Children's Hospital group in this regard are of interest to me, in that I am sure they are describing the same condition, although they do not use the term "neonatal hepatitis." They refer to this as inspissated bile syndrome of unknown etiology. Their experiences, otherwise, I think, have been comparable.

Dr. Norris has mentioned his feelings about Dr. Clatworthy's procedure, and I agree. If one does find the gallbladder and can make the study through a small incision, that is excellent and the operation can be terminated at this point. However, if the gallbladder is not present, I see no reason why one should not continue with the general exploration and dissection at that time.

The choleretic employed was magnesium sulphate or decholin,® or similar substances intravenously. We have not used cortisone in any of these cases.

The Repair of Large Omphalocele Defects—Simplified Method

HARRY E. PETERS, JR., M.D., *Oakland, California*

OMPHALOCELE is the descriptive term applied when an infant is born with an umbilical defect permitting the extrusion of abdominal contents into a sac formed solely of peritoneum and amniotic membrane.

This abnormality is uncommon, occurring once in every 6,000 to 10,000 births.

The size of the fascial defect in an omphalocele varies from a few centimeters to 15 cm. or more. In a small omphalocele the contents of the sac include a short segment of the intestinal tract. In a large defect, however, most of the intestinal tract, the liver and the spleen may be found in the sac.

In the normal fetus during the fifth to tenth week of intrauterine life, disproportion in growth of the intestinal tract compared to the abdominal cavity necessitates prolapse of the intestinal tract into the umbilical cord. By the end of the tenth week this disproportion is overcome and the abdominal contents return to the abdominal cavity, thus permitting closure of the defect in the anterior abdominal wall. If there is excessive growth of the intestinal tract, however, or inadequate growth of the peritoneal cavity, some of the viscera remain prolapsed into the umbilical cord. This arrests adequate development of the coelomic cavity, prevents closure of the abdominal wall and results in the development of an omphalocele.

The presence of an omphalocele, a most serious problem a few decades ago, now can be treated successfully, perhaps with the exception of the extremely rare "complete" omphalocele or in instances of intrauterine rupture of the sac. This improvement in treatment is the result of several factors. Of importance is the present appreciation of the urgency of prompt repair. If an infant is born with an omphalocele, repair must be carried out within the first four to five hours after delivery. Delay beyond this period invariably leads to peritonitis from bacterial invasion of the thin and fragile amniotic membrane.

An important contribution to therapy was made by Gross with his introduction of a two-stage method of closure for large omphaloceles. Repair of the small omphalocele, with a fascial defect of less than 5 cm., has posed no problem. Excision of the amniotic membrane with layer closure of the abdominal wall is most satisfactory. However, this technic is not applicable to the repair of large omphaloceles with a fascial defect of 8 cm. or more. Attempt at primary layer closure of these large defects, even if technically feasible, leads to a marked increase in intra-abdominal pressure with elevation of the diaphragm and pulmonary embarrassment, circulatory failure from portal and caval pressure, and intestinal obstruction. The ingenious two-stage method of repair as devised by Gross provides a most adequate solution. In the first stage of this procedure the amniotic membrane is left intact and the lateral skin flaps are mobilized for closure over the omphalocele. No attempt is made at fascial repair of the defect at this time. The second stage of definitive repair is carried out six to twenty months later when the abdominal muscles and fascia have stretched and adequate enlargement of the abdominal cavity has occurred.

Of significance, too, in our present successful therapy is a better appreciation of the importance of fluid and electrolyte balance, improved anesthesia and the judicious use of antibiotics in the management of the surgically treated omphalocele.

There can be little doubt that early primary definitive repair of the omphalocele, when possible, is the treatment of choice. When the size of the defect of the omphalocele requires a two-stage closure, a layer closure at the time of secondary repair is the best method. In some instances, however, the fascial defect is of such

magnitude that satisfactory layer closure may be both difficult and hazardous. It is in this particular group of cases, and this group only, that my technic is offered as a simple and effective method of repair.

No originality for the method is claimed.

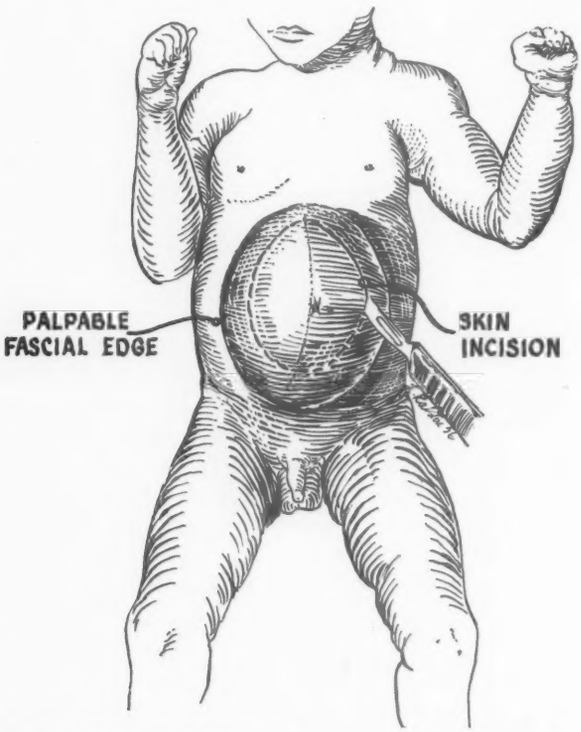


FIG. 1. A skin incision is made 1 to 1.5 cm. inside of the readily palpable fascial edge.

Similar principles have been employed in other surgical procedures, but apparently have not been applied to the repair of an omphalocele.

The method to be presented embraces the use of a cutis graft, removal of the superficial epidermis by a dermatome and mobilization of the lateral skin flaps for closure over the graft.

A circular skin incision is carefully outlined just 1 to 1.5 cm. inside the readily palpable fascial edge at the periphery of the defect. (Fig. 1.) This will produce the necessary tension in the subsequent repair to minimize cyst formation. The peripheral skin flaps are then mobilized sufficiently to permit closure, under slight tension, over the cutis graft. This exposes the fascial edge to be used in the repair. In the next step, the superficial epidermis ($^{6}/_{1000}$ to $^{8}/_{1000}$ inch) is removed from the graft by means of a dermatome, and this tissue is discarded. (Fig. 2.) This maneuver provides the advantage of a cutis or dermis graft over a full-thickness graft. It removes bacterial contaminants, eliminates subsequent slough of the epidermis and facilitates firm fusion with the fascia to which the graft will be sutured. Removal of the epidermis also promotes adherence between the dermis graft and the skin flaps which will be brought together over the graft.

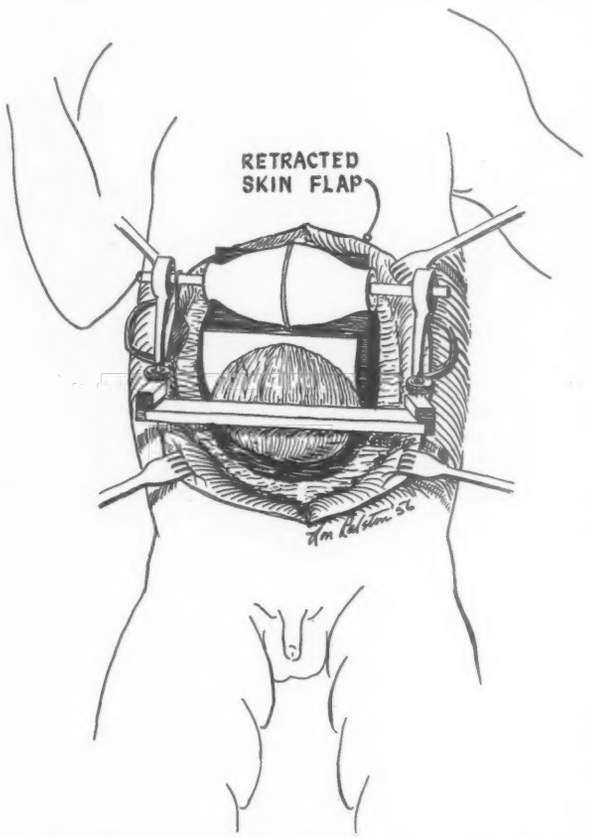
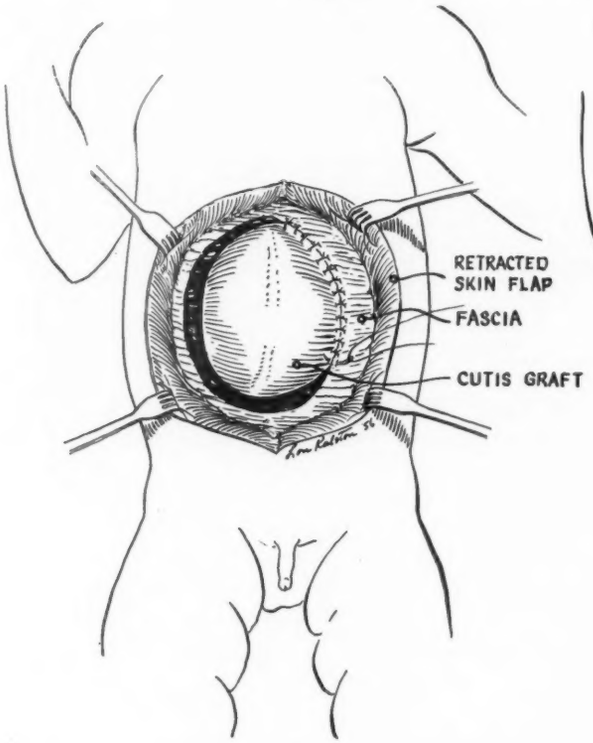


FIG. 2. The dissected skin flaps are retracted and the superficial epidermis is removed from the *in situ* dermal graft by means of the dermatome.

Since 90 per cent of the strength of a dermal graft lies in the dermis, the adherence of the graft to the overlying skin provides a double layer for additional strength. The periphery of the graft is now approximated to the adjacent fascial edge, using interrupted sutures of fine cotton or silk. (Fig. 3.) The procedure is completed by apposition of the skin flaps over the graft. (Fig. 4.) It may be desirable to place a small Penrose drain under each lateral skin flap and bring it out through a small stab wound. A soft compression-type dressing is then applied.

SUMMARY AND CONCLUSION

1. A simplified method is presented for secondary repair of those large omphalocele defects that may not otherwise be repaired successfully.



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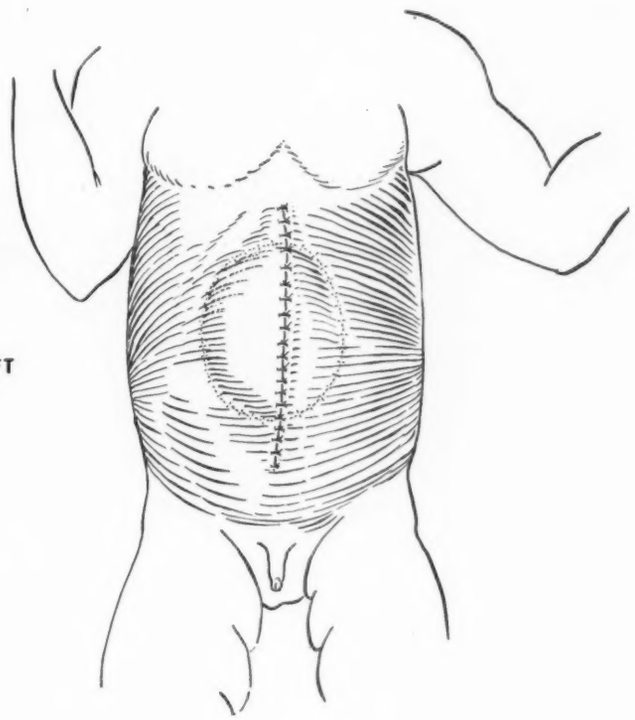

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FIG. 3. The periphery of the dermis graft is approximated to the fascial edge using non-absorbable suture material.

FIG. 4. Skin edges are approximated over the dermis graft repair of the defect.

2. This technic embodies the use of a dermis graft placed under tension to minimize cyst formation. The removal of the superficial epidermis by dermatome reduces bacterial contamination, eliminates slough and promotes firm fusion with the fascia.

3. Of significance with this method is the absence of any marked increase in intra-abdominal pressure, and hence a smooth and uncomplicated convalescence can be predicted.

4. With this type of hernioplasty the suture line is not subjected to the greatest stress at its weakest point.

5. The first patient, on whom the technic under discussion was employed, was operated upon in December, 1955. The result at this time is most satisfactory but further observation is necessary to determine the ultimate success of the method.

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DISCUSSION

DONALD BRAYTON (Los Angeles, Calif.): It is indeed a pleasure to discuss Dr. Peters' interesting paper. I think it only fair to say that in spite of improved technics of repair the mortality of infants with omphalocele remains extremely high. This is due to several factors, including the frequent association of this condition with other anomalies, both of the gastrointestinal tract and elsewhere, that are incompatible with life. Intrauterine rupture of the amniotic membrane or its rupture during delivery often defeats the surgeon, and the very large defects without adequate development of the abdominal cavity add further to the mortality per cent. We have checked the total death rate from this condition at Childrens Hospital in Los Angeles

during two recent time intervals. The improvement during the most recent of these periods represents to some extent the effects of the adoption of Gross' technic of primary repair. However, there are so many variables inherent in and with the condition that the mortality may fluctuate widely from year to year in spite of such technical advances.

Since the small omphalocele defects are the most favorable for repair, it follows that most infants who live after omphalocele repair have had a primary successful closure. Those infants surviving to be candidates for a secondary procedure constitute a relatively small percentage of the total group. For instance, in a rapid and necessarily incomplete survey of the majority of the cases seen recently at Childrens Hospital, only one of twenty-one patients eventually became a candidate for secondary repair. Although this secondary repair group is small, the successful eventual correction of their remaining abdominal wall defects at an early age is of great importance, since having survived the original operation and passed through early infancy, it may be presumed that these young children are essentially normal in other respects.

The technic for secondary repair described by Dr. Peters is most ingenious and seems to me to add considerably to the surgical armamentarium in approaching this problem. It has the advantages of imposing no direct surgical contact with the peritoneal cavity, and of interposing a tough reparative layer retaining its own blood supply over the defect under optimum conditions of tension. It does, however, have the disadvantages which may be associated with cutis grafts, such as serum formation, potential infection and stretching. The danger of infection may, perhaps, be kept to a minimum by meticulous cleansing of the skin to be buried during a period of one or two days preoperatively. The removal of $^{6}/_{1000}$ to $^{8}/_{1000}$ inch of epidermis with the dermatome should provide for adequate healing between the graft and the overlying subcutaneous tissue without unduly limiting the tensile strength of the graft.

I believe Dr. Peters' technic deserves a long and careful trial, and feel that in all likelihood it will attain a useful and important place in the secondary repair of omphalocele.

MILLARD S. ROSENBLATT (Portland, Ore.): I think Dr. Peters' paper is a significant contribution to the subject of large omphalocele defects.

I wish to sound one note of caution from the experience we have had with cutis grafts in the repair of large ventral hernias—a small experience, to be sure, comprising only eight cases. I do not believe it wise to employ drainage in these cases. The only case in which we used drainage is the only one with which we had trouble. If you do employ drainage, the drain should not be left for any length of time. The potential space between the cutis graft and the overlying skin is easy prey to infection, and despite the small thickness of skin that you may take off, there is still a possibility of keratohyalin debris being deposited there. The addition of a drain for any length of time to allow ingress of bacterial contamination may cause a very distressing postoperative complication.

Our longest experience with this type of circumstance, not an omphalocele but in a large ventral hernia defect, dates six years, and the only case in which we had any trouble was the one in which we left drains.

R. BRUCE HENLEY (Oakland, Calif.): I, also, would like to congratulate Dr. Peters for his very excellent presentation of an ingenious method of repair for large omphaloceles.

I would like to discuss the case of a two week old baby who at birth underwent primary skin closure of a large omphalocele. All the intestinal tract, a portion of the stomach and a small portion of the liver were visible at birth. When the boy was two years of age, the huge abdominal hernia was repaired through a long vertical incision through the lateral border of the rectus fascia on each side. On one side we turned a complete flap of the rectus fascia and sutured this to the relaxed fascia on the opposite side, allowing a small border for overlapping. This method gave us an additional 2 inches for closure of the abdominal wall. We could have gained more if we had turned the rectus sheath on the second side as we did on the first.

A preoperative chest plate showed the normal level of the diaphragms. A chest plate taken the day following surgery showed that the diaphragms had risen 50 per cent. There was evidence of marked respiratory distress with increased respirations and rapid pulse rate. Herein lies the contribution of Dr. Peters. His method of repair would obviate any possible respiratory distress.

On examination before the boy's hospital discharge the wound was well healed and the repair successful. Four years have passed since the repair. The patient was recently examined and there was no evidence of weakness or recurrence.

HARRY E. PETERS, JR. (closing): I would like very much to thank the kind discussers of my paper.

In regard to Dr. Rosenblatt's comment, I might add that if drains are used they certainly should not be placed adjacent to the graft but should be placed out laterally in the gutter.

Dr. Longmire has suggested a continuous suction technic comparable to that employed after surgery of the breast, and I believe this should work very well.

I might mention in closing, Dr. Gross of Boston, in a personal communication, indicated that they had encountered some difficulty at the second stage closure of large omphalocele defects. They are now treating these cases in multiple stages. Dr. Gross also mentioned that he was very much interested in the method presented in this paper.

The Management of Hypertrophic Pyloric Stenosis at the Los Angeles Childrens Hospital

A REVIEW OF 1,422 CASES

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CONGENITAL hypertrophic pyloric stenosis is the most common condition requiring surgical treatment in infancy. It occurs about once in every 300 live births in this country. The first American case was reported by Beardsley of New Haven in 1788. Our understanding of the pathologic anatomy dates from the classic studies of Hirschsprung in 1888. The mortality rate from medical treatment before surgery was attempted was about 80 per cent [*33*] and the use of gastroenterostomy reduced this to about 50 per cent [*18*]. In 1908 Fredet originated the longitudinal division of the hypertrophied pyloric muscle while leaving the mucosa intact, although he also attempted transverse suture of the muscle incision. Rammstedt in 1912 improved the operation by omitting any attempt at transverse suture of the cut muscle; he left the mucosa exposed with the muscle gaping. The success of the Fredet-Rammstedt pyloromyotomy is one of the most outstanding in all the practice of surgery. Many modern reports indicate that pyloric stenosis can be treated with this operation with a very low mortality rate. In the last twenty years at the Los Angeles Children's Hospital we have seen 1,422 patients with pyloric stenosis and the over-all mortality rate for the series is 1.7 per cent. This report is based on a detailed study of 312 consecutive cases from the last five years.

Etiology and Pathogenesis. The etiology of this disease is still unknown. Many theories of its cause have been offered, but none is entirely acceptable. Some of the theories are: (1) pylorospasm is the primary lesion followed by muscle hypertrophy; (2) muscle hypertrophy is the primary lesion followed by spasm; (3) imbalance of the autonomic nervous system with hypertonia of the vagi; (4) gastrointestinal allergy; (5) vitamin B deficiency in the mother during gestation; (6) pituitary lesions; (7) injury to central nervous system at birth; (8) myenteric ganglion cell degeneration or lack, analogous to Hirschsprung's disease; (9) neoplasm; (10) glycogen storage disease; (11) adrenal cortical deficiency or dysfunction ("dyscorticismus"); (12) mother elaborates a hormone which is "pylorotropic," in males leading to hypertrophy of pylorus; in females to hypertrophy of the uterus; (13) maternal emotional disturbances; and (14) anomalous blood supply to the pylorus.

None of the theories thus far advanced can satisfactorily explain three cardinal features. First, the fact that 80 per cent of the cases occur in males. Second, the long symptom-free interval between birth and onset of vomiting. The longest reported interval, in a proved case, is five months [*37*]. Third, the fact that as a clinical entity the disease is limited to the first few months of life. In adults with surgically proved pyloric stenosis the clinical syndrome is never that seen in infants.

Some investigators believe that hypertrophy of the pyloric muscles may be present for some time before the clinical symptoms become apparent [*9,33*]. It is thought that the efforts of the stomach to force thick curds through the narrowed canal causes the edematous mucosa to prolapse into the canal and obstruct it. This

may be accompanied locally by microscopic signs of inflammation. Relief by medical means is partially achieved by reducing the vigorous efforts of the stomach muscles proximal to the rigid pyloric canal, thus permitting the mucosa to return to its normal position and thickness. Dilatation and hypertrophy of the antrum may appear in delayed cases. The hypertrophy of the pyloric muscle is limited to the circular layers and the size of the muscle mass is proportional to the age of the patient [*26*].

In one reported case a No. 14 catheter passed the pylorus in an infant proved to have pyloric stenosis at surgery a few days later [*48*]. It is generally agreed that the pyloric obstruction is never complete, but the tiny lumen remaining is not adequate to maintain nutrition.

The muscle hypertrophy and deformity of the pylorus end abruptly at the duodenum, and the duodenum is entirely normal. Perforation of the duodenal mucosa can easily occur if the surgeon carries the myotomy too far distally.

Wollstein's classic study showed that after pyloromyotomy the muscle cells show no proliferation and the wound is healed by fibrous tissue by the ninth to thirteenth day after surgery although there is still a visible depression of the serosal surface. Further healing causes some contraction of the fibrous scar. By the twenty-fifth day there is only a narrow scar and by eighteen to twenty-four months the scar is almost invisible. This finding can be confirmed grossly in patients reoperated upon for incomplete myotomy. It has been shown that the pyloric muscle changes will persist into adult life in infants treated by gastroenterostomy [*3*].

Associated Anomalies. In our series 12 per cent of the infants with pyloric stenosis had some other unrelated disorder such as hernias, digit anomalies, cryptorchidism, polythelia, etc. Most of these were minor anomalies, but a portion of our mortality came from the coexistence of pyloric stenosis with other major congenital defects such as congenital heart disease, imperforate anus, esophageal atresia, etc. Other authors report an incidence of associated anomalies, of all types, of 2.7 per cent [*23*], 3.4 per cent [*31*] and 8 per cent [*17*]. It is generally agreed that there is no association of pyloric stenosis with any other anomaly of the infant or with maternal rubella [*23*].

Sex Incidence. This is clearly a disease occurring predominantly in males. The voluminous literature is in complete agreement that about 80 per cent of pyloric stenosis cases occur in male infants [*8,11,12,17,23,25,31,38,39,43*]. In our series exactly 80 per cent were in males.

There is no apparent racial predisposition. It has been suggested that this disease is uncommon among the Orientals and less common among Negroes, but this impression is not well documented [*31,33*].

It has been said that babies with pyloric stenosis tend to be larger than normal. Malmberg found that 60 per cent of his patients had birth weights in excess of 7 pounds, 10 ounces, but Corner found that the birth weights in his series paralleled almost exactly those of normal infants born in Bristol, England. In our series the average birth weight was 7 pounds, 7 ounces. This is only 4 ounces heavier than the average birth weight of white babies born in the state of California (1953 report). This slight advantage in birth weight is more than lost when growth curves of babies with pyloric stenosis at the average age of operation as compared with those of normal babies [*31*]. (Table II.)

Birth Rank. Despite statements to the contrary [*10,15,31*], it can be clearly shown that there is a definite excess of first-borns among cases of pyloric stenosis compared with their proportion in the general population. It has also been said that this preponderance of first-borns does not appear in the cases with onset in the first two weeks [*27*] (or three weeks [*6*]) of life. In our series this was not true. Birth rank bore no relation to the age at onset. The first-born preponderance is well shown by comparison with the normal population of California and the entire United States. (Table I.) More than one-half of the babies born in the state of California are born in the metropolitan Los Angeles area so our sampling is probably representative.

Family History: The Evidence for Genetic Factors and for Environmental Factors. The characteristic pathologic lesion has been found in a seven-month fetus, in stillborns, in newborns and in premature infants [*14,21,41*]. The presence of pyloric muscle hypertrophy in a non-viable infant does not prove that the infant would have had the clinical syndrome of pyloric stenosis. There are many proved surgical cases in newborns, and strangely most were in females. Green and Sidbury reported the case of a 6 pound female infant with vomiting from the age of six hours, typical gastric waves and

TABLE 1

INCIDENCE OF PYLORIC STENOSIS IN INFANTS, ACCORDING TO BIRTH RANK

Source	First-born (%)	Second-born (%)	Third-born (%)	Fourth-born (%)	Fifth-born, Plus (%)
McKeown (England)	51	29	12	4	
Corner (Bristol, England), 117 cases	50	25.6	12.4	8	5
Malmberg (Sweden), 136 cases	61	27	6.6	3.7	1.3
Rinvik (Norway), 137 cases	54	29	10		
Gerrard (England), 152 cases	35.5	31	18	8.5	6.6
Grimes (San Francisco), 119 cases	52	25	17	2.5	2
Donovan (New York), 245 cases	53				
Miller (Kansas), 74 cases	33.8	38	23	2.7	2.7
Los Angeles Childrens Hospital, 312 cases	40.6	30.4	18.2	7.8	1.9
California, 1953: Total white births (297,616)	31.1	30.2	20.0	9.7	4.3
U.S.A., 1953: Total white births (3,902,120)	28.3	27.5	18.5	10.1	5.3

a pea-sized tumor; she was operated upon on the third day of life. A small but definite tumor was found and the postoperative course was smooth. Kaiser reported the case of a 3 pound, 11 ounce premature male who began vomiting fourteen hours after birth. Visible waves were seen from the third day, and at operation on the fifth day of life a typical pyloric tumor was noted. The patient did well postoperatively.

MacHaffie reported on an eight pound female infant with a history of gagging and cyanosis coming from regurgitation of mucus immediately after birth. Vomiting increased after feeding began, and a barium study revealed pyloric obstruction; the barium was vomited. No waves were seen and no tumor palpated. Operation was performed when the infant was thirty-four hours old (the youngest patient ever operated upon) and a typical tumor was found. The child had a successful result from pyloromyotomy. Many children with pyloric stenosis will have gagging and occasional respiratory distress from the excessive amounts of mucus in the early days of life. Meeker and DeNicola reported the case of a female infant, weighing 8 pounds, 3 ounces, who began projectile vomiting thirty-six hours after birth. Temporary improvement was attained with atropine and thickened feedings, and no waves were seen or tumor palpated. Operation was performed on the fourth day of life and a typical pyloric stenosis found. The stormy postoperative course was characterized by the vomiting of bile (previously absent) but eventually the child recovered.

Kessler and Scott reported on two Negro infants who had had vomiting from birth. The female infant was first seen on the eighth day of life and not operated upon until the eighteenth day. A tumor was felt and waves observed. The child died, probably from aspiration of vomitus following a second operation three days later for bowel obstruction due to volvulus. The second child, a male, weighed 6 pounds, 13 ounces at birth and vomited after feeding was started twelve hours after birth. Operation was performed on the fifth day with typical findings and a prompt recovery. Rothman, Fields and Naiditch reported the case of a white female infant, weighing 7 pounds, 12 ounces at birth, who began regurgitating mucus from birth with gagging and cyanosis. Visible waves and a palpable tumor were noted from the fourteenth hour of life. Operation on the third day of life revealed typical pyloric stenosis, and recovery was prompt and complete.

In 4 per cent of Rinvik's series and 10 per cent of Miller's series vomiting started on the first day of life. In our series ten babies, of the last 312, had a history of vomiting from birth. Operation was performed upon one in the second week, three in the third week, four in the fourth week, one in the eighth week and one in the twelfth week of life.

There is a considerable body of evidence

favoring a genetic factor in the etiology of pyloric stenosis [*5,30,45*]. Both members of monozygous twin pairs were involved in 66 per cent (twelve of the eighteen pairs) compared with only 3.5 per cent (one of twenty-nine) involvement of both members of dizygous fraternal twin pairs [*30*]. The evidence offered from twin studies by Metrakos and by Sheldon has been strongly challenged on the grounds that the monozygosity has not been conclusively demonstrated and that the "penetrance" of the gene composition must be incomplete [*5,28*].

It has been said that the risk of pyloric stenosis is about one in ten for brothers or for sons of known cases of pyloric stenosis, and the risk for daughters or sisters of known cases is of the order of one in fifty. There is apparently no increased incidence of the disease in first cousins of known cases [*6,7,26*]. The latter point has been used to indicate that the genetic composition must be of less importance than the previously noted sibling-parent evidence would suggest.

The risk of pyloric stenosis is four times greater in children if the mother had had pyloric stenosis than if the father had had it [*28*]. Several instances of affected half-siblings from the same affected parent are reported [*26*].

It has been suggested that pyloric stenosis is more common in twins than in single births, but this is apparently not true. The incidence of twinning among pyloric cases is no different than the incidence among the general population (about one in forty births) [*5,30*].

Nielsen (Denmark) studied seventy-three males and twenty-two females aged twenty-five to forty-five who had all been medically treated for pyloric stenosis in infancy. As a control group he chose 100 unselected hospital patients, seventy-seven males and twenty-three females aged twenty-five to fifty-one. The control group had no gastrointestinal problems as their presenting complaint. The parents with pyloric stenosis (ninety-five people) had had a total of 128 children of whom eleven had pyloric stenosis, and the ninety-five parents had 216 siblings of whom nine had had pyloric stenosis. In addition, there were eight cases of pyloric stenosis in other relatives of the ninety-five parents studied. In contrast, the control group of 100 people had had 145 children of whom only one had had pyloric stenosis, and the 100 parents had 319 siblings of whom none had had pyloric stenosis. There was one case of pyloric stenosis in a more distant relative. Nielsen concluded that there is a considerable familial accumulation of pyloric stenosis and that the disease is partly hereditary in origin.

In Cockayne and Penrose's series of 212 cases, nine siblings of known cases had pyloric stenosis. In the University of California series (119 cases) two uncles, six brothers, one with two brothers, three cousins and one father had had pyloric stenosis [*17*]. In our series of 312 cases we had ten with involved siblings, three with involved parents, three with involved cousins and twelve with involved aunts, uncles or parental cousins.

The evidence favoring a postnatal environmental factor is equally interesting. It is an established fact that babies with pyloric stenosis do not start vomiting until feeding is started. The interval between birth and the onset of vomiting is amazingly long in some cases [*45,50*]. Rendle-Short noted seven cases with the onset after eight weeks, Corner reports one with the onset on the eighty-second day of life, and in Grimes' series one patient was first seen at five months of age. Rendle-Short had one case with the onset ten days prior to admission at the age of five and a half months of age, the longest interval found in the literature. The longest interval in our series was ten weeks (two cases).

Reports from England indicate that the onset of vomiting is later in babies born in the hospital than in those born at home. Some authors believe that experienced hospital nurses delay the onset of the serious vomiting as compared with relatively inexperienced mothers. Inexpert feeding may be a partial explanation for the preponderance of first-born children among pyloric stenosis cases. It has been shown that if the baby is fed every three hours, the onset is earlier (about twenty-one days of age) than if the baby is fed every four hours (about twenty-seven days). The onset of serious vomiting leading to hospital admission occurs after about 130 feedings on the average. The more often the baby is fed, the sooner he is admitted to the hospital. The acceleration of the onset of symptoms from three hourly feedings is even more marked in second- and third-born children than in first-borns [*15*]. There is a slight increase in the incidence of the disease in the months of April to June and September to October [*25,39*]. No explanation of this has been offered, but the

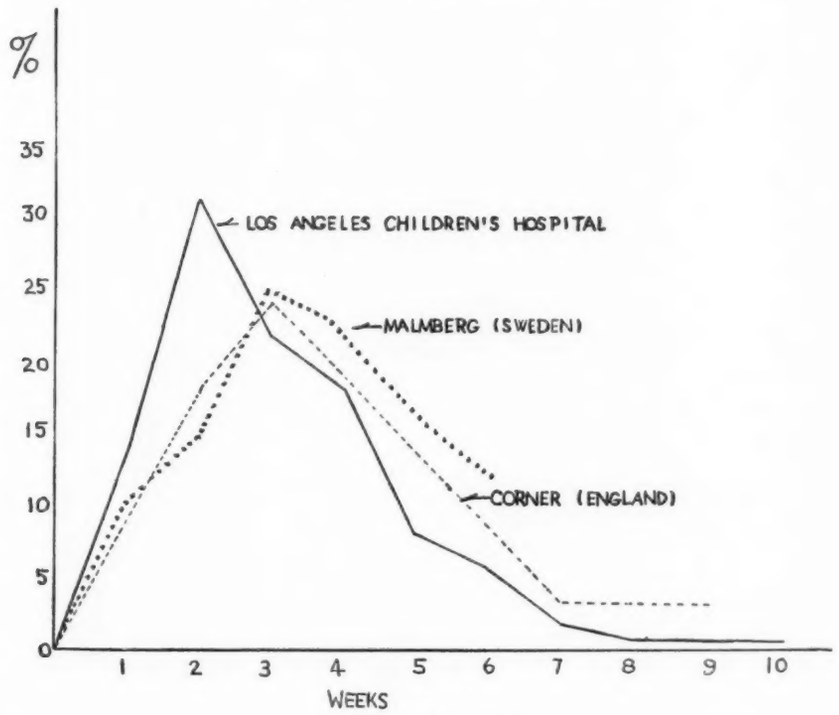

FIG. 1. Age at onset.

same seasonal increase applies to peptic ulcer in adults.

Many authors have referred to the study of Wallgren (Stockholm) who was able to obtain practically perfect follow-up studies on every male infant born within the area. The expected incidence of pyloric stenosis in male infants in Stockholm is about one in 150. He took routine gastrointestinal x-rays of every newborn boy in that hospital until he had accumulated a series of 1,000 cases. Five of these boys subsequently returned to him with x-ray evidence of pyloric stenosis and a typical history and physical findings. All of these infants were treated with scopolamine methylnitrate (scopyl) according to the standard Swedish method and recovered. None of the cases was confirmed surgically. The allegation that pyloric stenosis appeared in a previously normal pylorus depends largely on the differences between the newborn and subsequent x-ray examinations. Even Wallgren did not consider that proof conclusive.

Vomiting. Typically the vomiting starts as simple regurgitation of part or all of a feeding, but it soon becomes more frequent and forceful and finally "projectile." Some Scandinavian authors believe that in some babies the onset is almost abrupt, with the day and even the hour or the feeding of onset clearly defined [*24*]. The meaning of "projectile" vomiting is apparently variable. Some use the term to mean very forceful vomiting which throws the vomitus for a considerable distance and usually empties the stomach in a single large emesis. Others consider it to mean that the vomiting arrives as a projectile does, i.e., abruptly and without the "warning" of nausea or retching. Nicholson mentions the "worried" look of a child with pyloric stenosis who is about to vomit.

Typically, these babies are hungry feeders; they act starved and will feed almost at once after vomiting. It is almost unknown for the vomitus to contain bile. The incidence of gross blood has varied from 2.3 per cent [*31*], to 4 per cent [*38*] to 8 per cent [*24*]. In our series gross blood was noted in 3 per cent. However, the vomitus in an additional 10 per cent was dark brown or "coffee ground," and chemical tests on some revealed blood.

The average age of onset is variously reported as twenty days [*31*], twenty-one days [*8*] and twenty-eight days [*39*]. We have prepared a line graph of the ages at onset in our series and in two other reports. (Fig. 1.)

Swenson found the average age at admission to be about six weeks, with the average duration of symptoms before admission about two weeks.

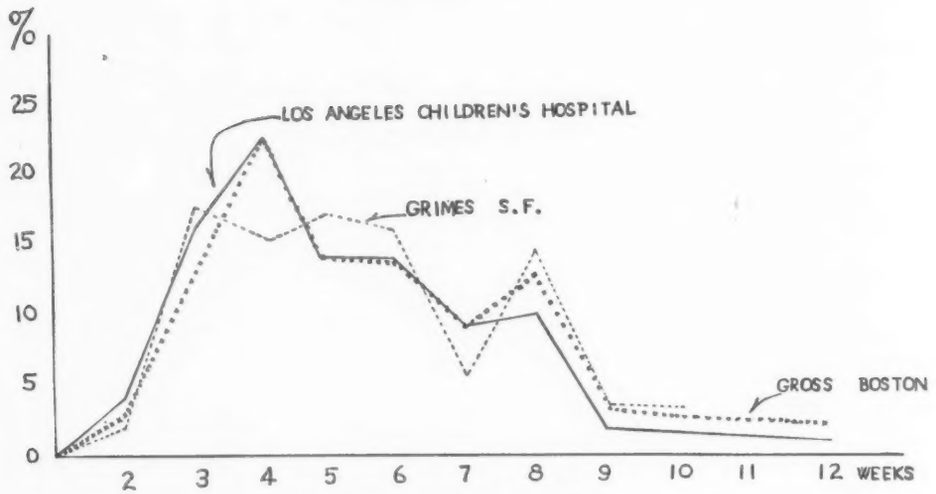

FIG. 2. Age at admission.

The average age at admission in Miller's series was 5.96 weeks, and the average age at operation in Savage's series was 4.3 weeks. The range was from one to eleven weeks of age. (Fig. 2.)

In our series the average age of patients with pyloric stenosis at admission in 1951 was 5.3 weeks. In the 1955 series the average age at admission was 6.2 weeks. We suspect that this shift toward later admission to surgical care means that longer trials of medical management are being used. A false sense of security is engendered by the fact that some of these babies will gain some weight on careful medical management. The idea is also current that surgery is not required if the infant can be carried past the sixteenth week.

TABLE II
WEIGHT GAIN: PYLORIC STENOSIS VS. NORMALS

Age at Admission* (wk.)	No. of Cases	Average Change from Birth Weight	Average Weight on Admission	Normal Weight for Age
2	19	6 oz. *loss*		
3	43	4 oz. *loss*		
4	62	4 oz. gain	7 lb., 12 oz.	8 lb., 8 oz.
5	42	13 oz. gain		
6	39	8 oz. gain		
7	29	16 oz. gain		
8	30	27 oz. gain	8 lb., 14 oz.	11 lb.
9	7	12 oz. gain		
10	7	21 oz. gain		
11	5	54 oz. gain		
12	7	35 oz. gain	9 lb., 1 oz.	12 lb., 6 oz.

* The ages at admission of our own cases and two other series are shown in Figure 2.

There is no doubt that the preoperative weight gain of the patients we have seen lagged far behind that of normal babies and behind that of babies with pyloric stenosis treated by pyloromyotomy at an earlier age [31]. (Table II.) The weight at admission in almost every instance was less than the maximal weight the baby had attained. We suspect that *this* weight loss rather than the failure to gain led to admission for definitive surgical treatment.

The dehydration and lack of food almost invariably result in some decrease in the frequency of stools. If "starvation diarrhea" supervenes, the situation rapidly becomes critical. The urine flow becomes scanty and concentrated. The babies usually fail to gain weight properly, or they actually lose weight [31]. Some believe that the combination of vomiting, dehydration, debility and electrolyte disturbances renders these babies prone to respiratory infections.

Physical Examination. One commonly noted feature of the physical examination is the presence of visible gastric peristalsis during the feeding. These gastric waves pass in regular fashion across the epigastrium from left to right. Grimes and Savage indicate that these waves were observed in "all" cases, Swenson found them "almost invariably," but Miller noted them in only 75 per cent. We have found them in 90 per cent of our cases. The presence of these gastric waves is *not* pathognomonic of

pyloric stenosis; they can occur in other totally unrelated conditions and in some normal babies [9,18].

One finding of great diagnostic significance is the palpation of a pyloric tumor. Craig states that it is possible to feel a pyloric muscle tumor and often to feel it contract under the examining finger in about 98 per cent of infants with pyloric stenosis over the age of two weeks. In children under the age of two weeks he found that in a series of 115 normal infants it was possible to feel a contractile pyloric tumor in sixteen males and in nine females, although none had any gastrointestinal symptoms. In a smaller group with recurring vomiting and failure to gain weight he was able to feel a contractile pyloric tumor in seventeen of twenty-one infants. Of these, three had sufficiently advanced signs and symptoms to warrant operation for pyloric stenosis. In two the pyloric tumor was small but definite and prompt relief followed pyloromyotomy. In the third, although a tumor was clearly palpated before surgery, the pylorus proved to be normal at surgery. In spite of the normal appearance of the pylorus and the absence of palpable thickening, pyloromyotomy was performed and the baby improved markedly. Craig explains the presence of a palpably contractile pyloric tumor in normal infants on the basis of a strong pyloric muscle spasm in the absence of hypertrophy. A brief tabulation of the reported experience from several centers is shown in Table III.

Nicholson noted that the tumor can sometimes be felt before the onset of projectile vomiting. In medically treated cases the tumor persists after complete clinical recovery [9,33]. It should be noted that the tumors in children under the age of two weeks are much harder to feel and are smaller both by palpation and at surgery. In about one-fourth of children under the age of three weeks with proved pyloric stenosis the tumor is not palpable at all. It has been shown that there is a direct correlation between the size of the tumor and the age of the baby at operation: the older the baby, the larger the tumor [26]. Donovan believes that the tumor can be palpated in every case and he does not operate until it can be felt.

Malmberg [24] takes a diametrically opposite view to that of Donovan. He says, " . . . examination for palpable or non-palpable pyloric tumor is completely unnecessary, unreliable, time consuming for the physician and unpleasant for the patient, whereas an x-ray examination gives the desired information rapidly, indisputably and conveniently." At the Childrens Hospital we have operated upon a small number of children with an otherwise typical history and physical findings even though the pyloric tumor could not be felt. We have found typical pyloric stenosis and muscle hypertrophy in all of these cases. Conversely, we have never operated upon an infant in whom a definite tumor has been felt only to find a normal pylorus at surgery.

TABLE III
PALPATION OF PYLORIC TUMORS

Source	No. of Cases	Tumor Definitely Felt (%)
Grimes	119	68
Rheinlander and Swenson	100	90
Akers	150 (1944–1948)	81.3
	150 (1949–1953)	74
Nicholson*		97.3
Rinvik	137	72
Svensgaard (Tallerman)	179	87
Savage	61	80
Craig (after age of two weeks)		98
Miller	86	80
Gross		95
Los Angeles Childrens Hospital	312	95†

* Tumor palpated in only 24.1 per cent in a series of cases in 1922–1933.

† In several of these cases the tumor was not felt at the admission examination but became palpable later during the hospital course.

In a few cases we have had the unfortunate experience, as most large teaching centers have, of opening an abdomen to find that vigorous efforts by inexperienced fingers to feel a small or deeply placed pyloric tumor have resulted in a subcapsular hematoma in the liver. This distressing finding means rough handling and we can only reiterate the prime necessity of gentleness in this palpation, as in all handling of these tiny patients. We have the impression that babies subjected to repeated deep palpation in this region have a more difficult postoperative course.

We have had a few patients treated medically

for a time in the belief that the infant did not have pyloric stenosis because the tumor could not be felt and the x-ray studies were equivocal. In our series six cases shown to have pyloric stenosis at surgery had had previous x-ray examinations which were thought to be negative for pyloric stenosis. This finding in the hands of expert radiologists may cast a shadow on the findings reported by Wallgren. The delay occasioned by the reliance on the x-rays and the inability to establish the diagnosis has been accompanied by a slight increase in hospital morbidity, but by no mortality.

Other centers have used diagnostic x-ray examination in 6 per cent [*18*], 19 per cent [*38*], 23 per cent [*17*] and 42 per cent [*43*]. In our series 22 per cent of the babies operated upon for pyloric stenosis have had barium studies of the upper intestinal tract. Of these, 14 per cent were done in the Childrens Hospital and the remaining 8 per cent elsewhere. We have not derived any reliable information about pyloric stenosis from plain films of the abdomen. A gas-filled stomach with *no gas* below this point must be extremely rare in true pyloric stenosis at the average age of this disease. In four cases barium enemas were given to rule out malrotation. We have had no untoward results from the vomiting or aspiration of the barium mixture. Some believe that watery suspensions of barium are not as reliable as barium mixed with formula in determining the degree of pyloric stenosis, but this question remains unanswered in the literature and in our experience.

It is generally agreed that retention of the barium in the stomach for three or four hours is suggestive but not diagnostic of pyloric stenosis. The finding of narrowing and elongation of the pyloric canal and decreased peristalsis occasionally occurs in infants who empty their stomach normally. The diagnosis of pyloric stenosis by x-ray requires the demonstration of pyloric deformity accompanied by evidence of gastric retention.

Akers did a particularly interesting study at the Denver Children's Hospital. In a series of 150 infants from 1944 to 1948 twenty infants had barium studies and eight had scout films. In a comparison series of 150 cases from 1949 to 1953 it was found that eighty-five infants had barium studies and two had scout films. The increased use of gastrointestinal x-rays to make the diagnosis indicated increasing confidence in the radiologist's ability to demonstrate the condition safely and reliably. Concomitantly the incidence of "palpable" tumors dropped from 81 per cent to 74 per cent. Akers believes that less vigorous efforts were made to feel the tumor after x-ray, as a diagnostic method, came to increased importance.

Corner states that of 117 infants treated with scopolamine methylnitrate, about 25 per cent were x-rayed before the start of treatment as part of a research project. Almost all of the infants were x-rayed after the completion of medical treatment. He found that the x-ray changes had returned to normal in all by two years after cessation of treatment.

In Caffey's first edition he said that "spasm of the antrum and stenosis of the pyloric channel may persist for years after surgical treatment and the complete relief of symptoms." In a small series studied by postoperative x-rays at this Childrens Hospital we cannot confirm this statement. In our cases the canal appeared essentially normal within a few months after surgery. Caffey's later editions do not include this statement.

In Scandinavian reports of cases treated medically and followed up by x-rays for many years, the typical pyloric deformity may persist up to the age of thirteen and occasionally beyond. Peristalsis does not visibly pass through the pyloric segment until about the second year of life although clinical recovery is complete [*2,25,35,39,42*].

Laboratory Work. In our series admission urinalysis has very commonly shown a trace of albumen and a "shower" of white cells with occasional other formed elements. This finding is linked to dehydration and clears promptly with rehydration. We have often found some acetonuria in the admission urinalysis. The pH of the admission urinalysis has been of no value in determining the state of acid-base balance. It bore no relation to blood chemistries. The average value for the series was exactly 7.0. The average admission hemoglobin value was 14.3 gm. per 100 cc. In those babies who had evidence of anemia after rehydration, small transfusions of whole blood or red cell sediment have been given. In our series 17 per cent of the infants were given blood during the preoperative or operative period. In Miller's series 60 per cent of the patients received blood transfusions. The average admission white blood count was 13,800 per cu. mm. and the differ-

ential counts, in general, followed the normal pattern for this age group.

We have routinely obtained values for carbon dioxide and chloride on admission; often a serum sodium and potassium and sometimes a non-protein nitrogen, blood urea nitrogen or other special test is made. In babies with marked disturbance of these values or serious fluid depletion these tests were repeated during the period of preparation for surgery. Electrocardiograms were taken on some with serious potassium imbalance. The *average* admission values in this series are shown in Table IV.

We have paid particular attention in this study to those babies who had the most marked disturbance of their chemical balances on admission to see if we could find a correlation between their clinical course and their chemistries. No consistent pattern could be shown. The babies were operated upon after a period of preparation which often appeared to be more dependent upon the restoration of fluid volume than upon the electrolytic components of the solution used. For example, some rather marked disturbances of the carbon dioxide and chloride levels were improved simply by giving glucose in water, but almost all of these infants required and received some electrolyte solution. The degree of dehydration was described as slight or mild in fifty-nine cases, moderate in sixty and severe in the remaining 193 cases. Ninety-nine per cent of this series were given some preoperative parenteral fluids. In Swenson's series 75 per cent and in Miller's series 100 per cent of the patients received parenteral fluids preoperatively.

We have usually used 2½ per cent dextrose in water, hypotonic Ringer's solution or half-strength saline for hydration. Swenson and Gross prefer the intravenous route. Other centers and our staff prefer the subcutaneous route, usually with hyaluronidase, if dehydration is not severe [*33,43*]. In this series of 312 cases the subcutaneous route was used exclusively in 172 cases and these babies received an average volume of 646 cc. preoperatively. In the remaining 140 cases both subcutaneous and intravenous or only intravenous fluids were used. The average total volume given these infants was 700 cc. Potassium chloride has often been added to these fluids as soon as the urine output was established. We consider it unwise to give this ion unless kidney function is adequate.

In the group of fifty-nine cases considered to have mild dehydration the average volume of fluid given was 523 cc. during the preoperative period. Infants with no evidence of dehydration require only maintenance levels of fluids and early surgery. Pyloric stenosis is not a surgical emergency and the days or hours spent in restoring fluid and electrolyte losses is time well spent. It might be suggested that suppressed renal function leads to retention of acid metabolites and consequently balances the alkalosis which results from protracted vomiting. Whatever the mechanism, the admission chemistries could not be related to the clinical course. The fact that the *average* values were essentially within the normal range came as a surprise to many.

TABLE IV

	Los Angeles Childrens Hospital	Normal Range
CO_2	27.4 mEq./L.	22–26
Chloride	97.5 mEq./L.	95–105
Sodium	138 mEq./L.	137–143
Potassium	4.3 mEq./L.	4.1–5.5

Almost all of our patients had had some antispasmodics and alteration of the formulas on the outside, but after admission they were taken off all oral feedings and therefore off the belladonna derivatives. We consider it dangerous to give a dehydrated, undernourished infant belladonna derivatives unless adequate amounts of food accompany it. The glowing reports from the Scandinavian and English literature about the use of atropine- or scopolamine methylnitrate have not been confirmed in a small series in our hands [*8,24*]. Our patients had been weaned to formulas almost routinely before admission. About 65 per cent of English babies are breast-fed through the age range in which pyloric stenosis occurs [*9*]. This may account for the English contention that pyloric stenosis predominates in breast-fed babies [*47*].

Anesthesia. Our anesthesia department is staffed entirely by full time medical specialists in pediatric anesthesia. This may account in part for the diversity between our methods and those of other institutions in this country and abroad [*11,44*]. Savage reports the routine use of open drop ether. They formerly used a preanesthetic dose of atropine but in recent years

have used no premedication at all. Swenson used open ether in ninety-five of the last 100 cases, cyclopropane in three and a local in two. Gross uses ether almost exclusively with only rare use of cyclopropane or a local. Grimes reported the use of general anesthesia in 105 of 113 cases with a local in eight. The British surgeons have a strong preference for the use of local anesthesia [44]. Our own methods are shown in Table v.

TABLE V
ANESTHETIC USED*

Anesthetic Used†	No. of Cases	Per cent
Ether	104	33⅓
Ethyl chloride and ether	69	22
Ethyl chloride, ether and nitrous oxide	31	10
Ether, cyclopropane and nitrous oxide	21	7
Ethyl chloride, ether, cyclopropane and nitrous oxide	19	6
Ether and nitrous oxide	19	6
Ether and cyclopropane	19	6
Ethyl chloride, ether and cyclopropane	11	3½
Local only	5	

* Supplemental oxygen was used throughout in all inhalation anesthetics (307 cases).

† The remainder had occasional use of vinethene,® pentothal® or other combinations of the above-named drugs.

It will be recognized that many of these babies had three or four inhalant anesthetics used in the induction or maintenance of anesthesia. As members of the surgical service we do not wish to give the impression that we are advocating this as a routine method for other hospitals. We are very well aware of the very great safety and other advantages of ether anesthesia with an oxygen stream under the mask for this operation.

The actual operating time for this series averaged thirty-three minutes; the range was twelve minutes to seventy-five minutes, the latter a case with mucosal perforation.

We no longer use a source of external heat as some other centers recommend [11,17,18,38]. We know of one baby (operated upon at another hospital) in whom use of a hot water bottle caused a third degree burn of the buttocks. We have checked the temperature of babies during this operation with continuous readings from a rectal thermocouple and find that if the baby has adequate covering and is protected from draughts, the body temperature does not drop significantly. This protection is important in air-conditioned operating rooms. Some surgeons prefer to warm the solutions used for the skin preparation and to avoid the use of agents which evaporate rapidly, e.g., ether, to avoid the cooling effect of this cold bath. We have usually used watery materials, e.g., hexachlorophene or zephiran.®

A small catheter, No. 12 or 14 French, is passed into the stomach preoperatively to empty it and is left on suction for twenty-four hours after surgery if the duodenum is perforated. Otherwise the tube is removed upon completion of the procedure. Some surgeons have used the tube to force air into the stomach after the myotomy to check for bubbles at the duodenal angle and this may be a useful maneuver, although we do not do it routinely. We have had no experience with the use of colored solutions, e.g., methylene blue, for this purpose or with the dubious maneuver of threading the stomach catheter through the pyloromyotomy to determine patency.

Incision. Gross states that for over ten years they have abandoned the use of right rectus incisions and prefer the Robertson incision. Swenson's group used the Robertson incision in 80 per cent, a direct transverse incision through skin and rectus in 13 per cent and a rectus incision in 7 per cent. In Savage's paper the "majority" were done through a Robertson or a transverse skin incision with retraction of the rectus muscle from the midline. Other centers much prefer the rectus-splitting incision and have used it in the majority of their cases [11,17]. In our series a rectus-splitting incision was used in 75.3 per cent of the last 312 cases, a Robertson incision in 10.6 per cent and a transverse incision through all layers in 10.3 per cent. In the remainder (3.8 per cent) the rectus was retracted from the midline, usually through a transverse skin incision. We have almost routinely used a layer closure of silk or cotton. We have had two instances of wound disruption, both in right rectus incisions. One in 1951 had a 1 cm. fascial defect which was closed. This child subsequently underwent a second myotomy at this hospital for persistent vomiting. The other baby died in 1943 following the secondary closure. In one other infant closure was effected primarily with retention sutures because he was under treatment with

adrenal cortical steroids at the time. We have found no cases of incisional hernias in the follow-up clinic.

We have found that in some babies with large tumors the Robertson incision gives rather limited exposure, and delivery of the tumor requires much more manipulation than a rectus incision. The rectus incision we use starts one fingerbreadth below the costal margin so that the liver acts as a buttress beneath it on closure.

We have routinely used gauze and tape dressings although occasionally the need for physical examination of the abdomen after surgery has made it necessary to remove the dressing. We have had very little experience with leaving the wound exposed, as recommended by Swenson.

Surgical Complications. Swenson reports that duodenal perforation occurred in 10 per cent of their cases, all in operations being performed by the resident staff. Savage reports an incidence of duodenal perforation of 6.6 per cent and Miller of 10.3 per cent. In six cases of the last 312 at this hospital the duodenal mucosa was perforated and closed without sequelae. In 1934 one death occurred with a localized peritonitis which may have been due to an unrecognized perforation. Pasma (Netherlands) states that some Dutch surgeons deliberately open the duodenal mucosa to be sure that all of the pyloric muscle fibers are divided. The increased complications resulting from this maneuver render it unacceptable to us.

Rinvik reported that in two of 137 cases incomplete myotomy was performed. Savage reported one in sixty-one cases, Miller one in eighty-seven cases and Gross noted four in the last report from his hospital. We had five cases of incomplete myotomy, three originally operated upon at this hospital and two operated upon elsewhere and referred to the Children's Hospital for further care. One of the two from another hospital was admitted in severe dehydration and electrolyte imbalance from the continuing vomiting, and died while being prepared for reoperation. The interval between operations in those operated upon twice varied from fifteen days to four months. All infants improved following the second myotomy. Gross inspection revealed that in all of them the healing process had completely covered the mucosa.

The literature suggests that bleeding from the cut muscle can be ignored [*38,44*]. We do not agree with this view. We had one death in 1937 from bleeding from the myotomy, and Miller reported one death in his series from this cause. In 25 per cent of our cases (seventy-eight of 312) we have taken the precaution of ligating or suturing small bleeding points in the muscle. In two of these the surgeon performed a second myotomy after several hemostatic sutures had somewhat compromised the lumen of the first myotomy. Miller reports significant oozing from the cut muscle in twenty-eight of eighty-seven cases (32 per cent).

We have had one rather unusual complication. The gastric mucosa was perforated in one infant; this was promptly recognized and sutured with fine catgut. The immediate postoperative course was complicated by minor hematemesis but on three subsequent occasions, at about monthly intervals, the infant was readmitted with more severe hematemesis requiring transfusion. No clinical or x-ray evidence of pyloric obstruction was noted and recovery thereafter was uncomplicated. We found no other instance of this particular complication in the literature. Our experience confirms the statements in the literature that in about 3 per cent of operations for pyloric stenosis some wound infection develops [*17,31,38,39,43*].

Postoperative Feeding. There are some interesting differences of opinion about this point. Some authors prefer to have the baby lie flat on his back during feeding; others prefer to have the infant held by the nurse [*11,17,18*]. Savage starts feedings four hours postoperatively, Gross starts four hours postoperatively, Swenson three to four hours postoperatively, we begin at six hours, Grimes at eight hours, Miller at twelve hours and Faber sometimes not until twenty-four hours.

Savage starts with water, Miller with water, Swenson with sugar in water, Gross with 5 per cent sugar in water and Grimes with formula low in fat. We start with amigen® in 5 cc. amounts and use 5 cc. increments. Savage begins with 4 to 8 cc. per hourly feeding and gradually increases this to 30 cc. per hour by the next morning. Swenson's group starts evaporated milk formula after twelve hours, increasing this rapidly to 30 cc. every two hours and full feedings by forty-eight hours. Grimes uses 4 cc. every two hours with 4 cc. increments every two hours and 2 cc. of elixir of phenobarbital with every other feeding. The initial feedings are done with an eyedropper until

30 cc. per feeding is reached, when the baby goes on the bottle. Gross starts with 1 ounce feedings and increases these to 15 ounces per day by the fourth day. We increase the amigen feedings 5 cc. every two-hour feeding for the first twenty-four hours and switch to formula at 20 cc. per two hours, increasing this through the next twenty-four hours to 30 cc. and then 45 cc., reaching 60 cc. of formula every three hours by forty-eight hours postoperatively.

Nicholson's group reports that in their cases 55 per cent had no postoperative vomiting, 18.6 per cent vomited for one to four days postoperatively and 20.6 per cent vomited for five or more days after surgery. Swenson's group reports that 60 per cent of their patients had no vomiting postoperatively and 35 per cent vomited only once.

In our series only 10 per cent had no regurgitation postoperatively while 54 per cent vomited less than a total of seven times. The remainder had more persistent difficulties for several days. The maximum number of times a baby vomited in this series was fifty-four. We have the impression that if gastric waves are seen postoperatively serious consideration should be given to a second myotomy. If at all possible, secondary myotomy should not be undertaken in less than two weeks. By this time almost 100 per cent of patients operated upon for pyloric stenosis will have stopped all vomiting.

Other centers report an average postoperative hospitalization of about five days; our experience is the same. The total hospitalization in this series averaged eight days. Some centers report longer postoperative hospitalization, e.g., Nicholson reports nine days and Miller, ten days. The risk of hospital cross infection in infants is a well known problem; there is some virtue in early discharge of thriving children.

Antibiotics were used postoperatively in sixty-two cases, the usual indication being infections of the respiratory tract. Vitamin preparations were used in six cases. We have not used Gross' routine of ascorbic acid postoperatively to promote wound healing.

Some parenteral fluids were used postoperatively for maintenance in 200 cases; the average volume was 400 cc. One hundred twelve patients had no parenteral fluids postoperatively.

Antispasmodics were used postoperatively in seven cases. Adrenal cortical supplements were used in three cases. We have been concerned about adrenal cortical function because of our mortality experience. There are some references to the problem in the literature [32,40].

Mortality. Deaths in our series occurred from a variety of causes, some of which have resulted in a reappraisal of our methods of handling these cases. One infant died three days after operation for esophageal atresia and was found at autopsy to have pyloric stenosis. One infant had been operated upon on the day of birth for imperforate anus and also had a dextrocardia. The baby died after operation for pyloric stenosis 21 days later under local anesthesia. The circumstances of this death raised the suspicion of adrenal insufficiency. Another infant who had been slow in restoring his fluid and electrolyte imbalances, in spite of adequate replacement, died after surgery and also had some evidence of adrenal insufficiency. Five of the infants who died had evidence of bronchopneumonia at autopsy, and one in whom autopsy was not obtained had atelectasis and pneumonia clinically. We have previously noted the death in 1934 from peritonitis possibly due to an unrecognized duodenal perforation, the one in 1937 from hemorrhage from the myotomy and one in 1943 after secondary closure of an evisceration. In one infant with continued vomiting, autopsy revealed aberrant pancreatic tissue in the duodenum and in the pyloric muscle, although death was attributed to atelectasis; the child had an incidental patent ductus arteriosus. Two other infants with multiple congenital anomalies were seen. One had a colostomy for imperforate anus and died the following day; autopsy revealed an unsuspected pyloric stenosis. The other had chalasia of the esophagus and vomited and aspirated repeatedly, finally dying of bronchopneumonia. Unsuspected pyloric stenosis was found in addition to the other anomalies. One death occurred when a severely anemic baby with pyloric stenosis was given a direct transfusion with the mother's blood. The baby died after only 15 cc. of blood had been given, although all of the cross matches were repeatedly shown to be compatible. We wonder now if this was an Rh incompatibility, although the Rh problem was unknown at the time this occurred. Two babies died seven hours and twenty-four hours after surgery, respectively, apparently from cerebral edema; one might have been related to overhydration. One infant died from purulent meningitis with bilateral purulent otitis

media, one died from extensive bilateral suppurative parotitis and another from diarrhea. The first two of these had pyloric stenosis demonstrated at autopsy and the third died of diarrhea after pyloromyotomy. One infant died early in this series when a 1 L. bottle of fluids was connected to a cutdown and allowed to run until empty due to a misunderstanding. Autopsy information could not be obtained for the remainder of the deaths.

TABLE VI

Year	No. of Cases	No. of Deaths
1934–1946	647	17
1947	122	2
1948	86	0
1949	80	0
1950	69	2
1951	101	2
1952	90	2
1953	71	0
1954	79	0
1955	77	0
Totals:	1,422	25 (1.7%)

Our results in the last twenty years, including every death in an infant with pyloric stenosis, regardless of the cause of death, are shown in Table VI.

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DISCUSSION

H. Glenn Bell (San Francisco, Calif.): The authors are to be congratulated upon their presentation of this material. Their report shows what can be done when there is close cooperation between the departments of pediatrics, surgery and anesthesiology. Care of these children is a community effort, in which no one individual or department should be too dictatorial. The excellent care these children received is reflected in the extremely low mortality rate and relative lack of postoperative complications in the authors' series of patients.

There is very little, if anything, that one could disagree with in this paper. From July, 1930 to July, 1956, at the University of California Hospital in San Francisco, we operated upon 141 patients with congenital hypertrophic pyloric stenosis; there were no postoperative deaths. There was one set of identical twin boys in this group, and this, incidentally, was back in the days when we often saw these children arrive at the hospital in a very perilous or severely dehydrated condition. Each of the twins, after being well hydrated under the direction of the pediatrics department, was operated upon under local anesthesia and given a sugar tit to suck on.

Fortunately we are seeing these patients at a much earlier stage today, and it is practically routine with us to use open drop ether with oxygen. I personally do not like the use of the shotgun type of anesthetic agent in these little tykes.

Several years ago we were doing studies on eye muscle and were interested in comparing hypertrophied eye muscle with the hypertrophied muscle of pyloric stenosis in infants. At the time of surgery, after we had made the usual longitudinal split through the muscle, we would make an elliptic incision and take out a crescent-shaped piece of muscle about $\frac{1}{8}$ inch thick for pathologic study.

The head nurse of the Pediatrics Department did not know we were doing this and asked if we were doing something different from usual. She had noticed that many of these patients did not vomit postoperatively, or if they did, they did not vomit as often as patients on whom we had performed the ordinary Fredet-Ramstedt pyloromyotomy. To me, this simply indicated that all of the fibers of the pyloric muscle were being cut.

As the authors have stressed, it is important to be certain that the mucosa has not been perforated. This is a very simple thing to do: some of the gas from the stomach is squeezed through the pylorus before the pylorus is dropped back into the abdomen and the abdominal wall closed.

Donald T. Hall (Seattle, Wash.): I have enjoyed this paper, and I believe that no one can take issue with the authors after looking at their mortality results.

I have not analyzed the cases at the Children's Hospital in Seattle, but to my knowledge we have lost about three of these babies in the last ten years, so I think we can perhaps at least approximate the authors' results.

I would like to ask one question, and that is with regard to breast feeding. It has been my clinical impression, which I have not substantiated from a review of the records, that many of the children

in whom this condition develops are those who have been breast-fed for a period of two or three weeks and are then transferred to cow's milk, or whichever feeding formula the pediatrician recommends. I am wondering if Dr. Pollock has correlated anything insofar as that particular factor is concerned?

Just one or two other points: Again, as many of you know, I have been slightly sensitive about the unnecessary use of transfusions, and I am wondering, in the authors' cases, if there was really a necessity for giving transfusions to 17 per cent of these babies? I presume that there might have been years ago, but at the present time the pediatricians and the medical population at large are much more susceptible to early diagnosis, and we rarely see these babies now in the dehydrated semi-moribund state that we did even ten or twelve years ago.

In regard to x-ray diagnosis, the so-called "string sign" and the delayed gastric emptying, I believe that the diagnosis clinically is usually quite adequate, without the necessity of resorting to the use of barium or lipiodol, whichever one prefers to use. Furthermore, we think it can be a rather misleading procedure, for we have seen several cases in which barium has traversed the pyloric obstruction very adequately and rapidly; yet with clinical symptoms persisting, operation revealed a very definite clinical pyloric tumor.

Perforation of the duodenal mucosa, although not desired, is certainly far from being catastrophic, provided it is recognized. I think it is the lack of recognition of this particular factor that is all important, as it is very readily taken care of by simply suturing a small bit of omentum into the opening. We have had no complications as a result of this maneuver.

WILLIAM F. POLLOCK (closing): Certainly Dr. Bell is entirely correct that this is a community effort, and it represents an enormous amount of hard work on the part of the pediatric staff and the anesthesia staff, in addition to the surgical staff.

We do not wish to advocate this "shotgun" anesthesia. We are very well aware of the great advantages of ether anesthesia

In reply to Dr. Hall's questions, in years gone by some of the men who were dealing with this disease used to have a battery of wet nurses all around the Children's Hospital, and they were brought in to feed these babies during the immediate postoperative period.

We have had no statistical analysis of the number of babies now being breast-fed, or those in whom symptoms developed when they were weaned.

Almost all of the babies with pyloric stenosis admitted to our hospital have been weaned to an artificial formula by the time we see them.

The British have enormous faith in breast feeding. They have enormous faith in local anesthesia. They use a much smaller volume of parenteral fluids preoperatively, and these are the three differences between their methods and ours.

The other difference, which distresses them, is their mortality rate, which ranges from 5 to 10 per cent.

We agree with Dr. Hall that we probably gave a higher percentage of transfusions than we should have. Why these babies are transfused is not always clear from the charts, and certainly the addition of foreign cells to babies of this age has a certain danger.

With regard to whether x-rays have been used too often, we believe that the palpation of the tumor establishes the diagnosis, and x-rays are probably not necessary in most cases.

In 2 per cent of our series x-rays were considered to be negative in patients subsequently shown to have pyloric stenosis. We are aware that all over the country there is increasing use of x-ray to make the diagnosis, and as this occurs a simultaneous decrease in the frequency of palpating the tumor has been reported.

I should like to thank the two co-authors on this paper, Dr. William Norris and Dr. Earl Gordon, the surgical resident at Children's Hospital.

We should like also to express our gratitude to the other general surgeons on the staff of Children's Hospital who have cooperated so much in this survey, and to all the pediatricians and other men on the staff who have cared for these children.

Enteric Reconstruction of the Esophagus in Children

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LESIONS of the esophagus preventing normal oral alimentation have challenged the imagination and skill of the surgeon for many years. Recently valuable and fundamental contributions for the ultimate solution of this problem have been advanced. At the present time a variety of procedures are available for the repair or substitution of part or all of the diseased esophagus. Still there remains considerable confusion as to which procedure or combination of procedures merit preferential consideration.

In benign esophageal lesions, the question of selecting the proper reconstructive procedures is of paramount importance. Clinical evaluation of early as well as late results is worthy of our critical attention.

In general, caustic burns of the esophagus have responded well to modern supportive treatment and careful dilatations. Congenital anomalies of infants have yielded well to direct surgical attack, so that esophageal replacement is seldom indicated.

It has been our policy to resort to esophageal replacement only when less radical methods of repair have failed or in situations in which normal oral alimentation is otherwise impossible. The relative merit of a given procedure therefore depends upon certain considerations. It is especially desirable to employ reconstructive methods which will permit normal nutrition, avoid serious cosmetic defects, and eliminate reflux ulceration and recurrent stricture formation. The history of the development of esophagoplasties has recently been reviewed adequately by Fry [1], Moroney [2], Oschner and Owens [3], Yudin [4], and others. Our experience with certain of these procedures in children is the basis of this report.

Esophageal replacements have been accomplished successfully by utilizing stomach, jejunum, colon and skin tubes to bridge the defects. Less successful have been the attempts to use plastic prostheses. The stomach has proved a satisfactory substitute for many cases particularly those in which the lower esophagus has been removed, and in a few cases in which a total or subtotal esophagectomy has been performed. There are, however, certain disadvantages in using the stomach for such purposes. In spite of all apparent precautions esophagitis and stenosis of the esophagogastric junction will occasionally occur. Respiratory function frequently will be reduced by the distended thoracic stomach. The danger of an insufficient blood supply to the fundus following extensive gastric mobilization leading to necrosis and disruption of the esophageal anastomosis, must not be regarded lightly. The incidence of this usually fatal complication seems to be greater in children because of the relative smallness of structures which may strangulate or be deprived of an adequate blood supply in the early postoperative swelling stage of healing. On occasion the stomach may not be available for extensive interpositioning because of existing disease or previous surgical alterations.

In the past four years at the Children's Orthopedic Hospital in Seattle, we have had occasion to reconstruct the esophagus in three infants. In each instance a different procedure was employed partly by design and partly because of extenuating circumstances.

The first case to be presented is that of S. D., who at the age of seventeen months ingested lye but was not hospitalized until six weeks after the incident. When seen at this time she was unable to swallow solids and could swallow only limited amounts of liquids. A Stamm gastrostomy was performed shortly after admission to the hospital. Dilatations commenced and these continued one to two times a week

both by mouth and retrograde through the gastrostomy. Despite frequent and apparently adequate dilatations, the esophagus became increasingly difficult to dilate. It was obvious after ten months that no improvement had taken place. The patient was unable to swallow soft foods and regurgitated most of her saliva. She was referred to the Thoracic Clinic for consideration of surgical treatment. The cervical esophagus had responded well to dilatation but the entire thoracic esophagus was scarred and stenosed. It was decided that the thoracic esophagus should be resected and an esophagogastrostomy performed after transplanting the stomach into the thorax. In October 1953, at the age of twenty-seven months, the child underwent a transthoracic esophagectomy with considerable difficulty due to the periesophageal scarring. The diaphragm was opened, the old gastrostomy was repaired, and the stomach mobilized along the greater and lesser curvatures so that adequate length was obtained. The esophageal opening was closed and a new opening in the fundus fashioned and sutured to the proximal esophageal segment. The diaphragm was sutured about the lower part of the stomach loosely after first performing a pyloromyotomy. The chest was closed with suction drainage. The postoperative course during the first few days was moderately precarious. The patient started to improve on the third day and continued to gain strength over the next few days. On the sixth postoperative day jaundice developed; this progressed in intensity during the next forty-eight hours, and she became listless and toxic. Her condition deteriorated and the next morning she was comatose. She died shortly thereafter on the eighth postoperative day. A thoracentesis prior to her death revealed foul smelling serosanguineous material presumably the result of an anastomotic breakdown. A postmortem examination disclosed that gangrene of the stomach with rupture was the primary cause of death. Only the lower one-third of the stomach was viable. The remaining fundic portion of the stomach was completely gangrenous above the level of the diaphragmatic opening. The diaphragm did not seem to constrict the stomach, but there was evidently sufficient pressure or compression to produce ischemic necrosis above this level. This unfortunate set of circumstances made us reluctant to attempt a similar procedure again in infants requiring extensive gastric mobilization.

The jejunum has certain advantages as an esophageal substitute in that it is of proper size, and should assist deglutition by peristaltic action. Many authors, including Yudin [*4*], Roux [*5*], Herzen [*6*], Reinhoff [*7*], Robertson and Sargeant [*8*], Harrison [*9*], and Longmire [*10*], have reported notable success with jejunal transplants. Very often, however, the use of the jejunum is not possible because of inability to mobilize a sufficiently long segment with adequate blood supply to bridge the defect. Gradual interruption of jejunal blood supply by multiple stage procedures has been described by Shumacker and Battersby [*11*], and Harrison [*9*]. Although successful on occasion, failures have occurred because of thrombosis of collateral vessels and constricting adhesions.

The most satisfactory location for jejunal transplants has been the subject of much discussion. The subcutaneous antithoracic route is regarded by many as the safest, but cosmetically and functionally it leaves much to be desired. When placed substernally, as Robertson and Sargeant [*8*] have advocated, there are many obvious advantages and few disadvantages. The posterior intrathoracic location has few advocates at the present time. The danger of vascular complication with extensive grafts in this location is a sobering deterrent. In addition, the magnitude of surgery necessary to effect an intrathoracic replacement in infants should not be minimized. Certainly, whenever possible a less formidable procedure is favored.

The advisability of anastomosing the distal end of the isolated intestine to the stomach has been discussed by Yudin [*4*], and Leven and Varco [*12*]. In infants and children we agree with those who believe nutrition is more adequately maintained if the stomach is not bypassed. In adults the use of a Roux-Y has had a considerable following in the past. There is, however, renewed interest in maintaining normal gastrointestinal continuity. With either procedure there is inherent danger of peptic ulceration. As Yudin [*4*] has suggested, one may be forced to convert one procedure into another if conditions so dictate.

The second case I wish to present is that of N. V., a girl fourteen months of age when first seen in the Thoracic Clinic. She had been referred to Children's Orthopedic Hospital from Dornbecker Hospital in Portland, where she was originally treated for a tracheoesophageal

fistula. A repair of a type 3 fistula had been attempted on the first day of life, but was impossible because of insufficient esophageal length. Therefore, the proximal pouch was brought out the left side of the neck and the distal segment was tied off at the site of the tracheoesophageal fistula and a gastrostomy was performed. The tracheoesophageal fistula (to the distal segment) recurred so the distal esophagus was transected at the cardia through a left upper abdominal approach, and both ends were closed separately. The mother had been faithful about giving routine gastrostomy feedings, for the child was in an excellent state of nutrition, but it was apparent to us that because of the discharge and maceration of the neck opening and the need for constant attention to the gastrostomy feedings, the child was being rejected by the parents.

In Thoracic Clinic it was decided to proceed with a substernal jejunal replacement leaving the blind segment of esophagus *in situ* for the time being. This was accomplished in two stages. The first stage was carried out on May 9, 1955, when the child was two years of age.

The abdomen was opened and a limb of jejunum was isolated from the bowel using the technic of Yudin [4]. The proximal limb was brought out the neck after tunneling under the sternum. The jejunal graft with its blood supply was passed behind the colon and stomach through the gastrohepatic ligament and then up to the neck substernally in an isoparastaltic manner. The distal end of the graft was anastomosed to the fundus of the stomach near the cardia. The jejunum was re-anastomosed and the abdomen closed. At the termination of the operation the color of the protruding segment of jejunum in the neck was satisfactory, but forty-eight hours later it was black and gangrenous. The abdomen was opened and the jejunal segment was withdrawn for inspection. The distal end only was involved so after careful manipulation additional length was obtained and the segment was replaced as before. One month later the second stage was carried out and the esophagus and jejunum were freed and a direct end to end anastomosis established.

The child has done well since oral alimentation has been achieved. On only one occasion has there been any regurgitation and this was of undigested food. The present result should be classified as functionally satisfactory.

The disadvantage of using jejunal grafts is that frequently it is extremely difficult or impossible to preserve a functioning segment of bowel. Mes [13] has stated that in his opinion more patient undergoing jejunal transplants perish than survive. Yudin [4] stated, in his often quoted article, that the total jejunal plasties can be very difficult, very tricky or absolutely impossible. In cases in which the jejunum was not suitable for reconstruction it was logical that the colon should be considered. During the past twenty to thirty years many authors have suggested its use. Some have advocated the right colon while others have used the transverse and descending colon. It seemed to us that the right colon would lend itself more readily for transplanting than any other particular segment and that the blood supply is constant and of good quality. The mesentery of the colon is thin and does not cause plication as is so characteristic of the small bowel. The importance of these facts is realized when the colon is mobilized, for each centimeter of colon freed is readily available for substitution. There is occasionally some variation of the blood supply to the right half of the colon, but it would be rare indeed to find a segment with adequate vascularity that could not be mobilized. If there was any question of an inadequate blood supply from the midcolic artery, the right colic artery could be ligated in continuity two to three weeks prior to mobilization and transplantation of the right colon.

Our third patient, S. M., was first seen at the Children's Orthopedic Hospital in October 1952 at the age of two years. Six weeks prior to admission he had ingested nitric acid, had received emergency treatment at another hospital and was discharged. Five weeks later it was apparent the child was having progressive difficulty in swallowing. At this time he was unable to take even liquids by mouth, and therefore he was referred to Children's Orthopedic Hospital for care. Shortly after admission a Janeway gastrostomy was performed and dilatations were begun. Twenty-four hours after the first dilatation the patient became febrile and signs of mediastinitis developed. The area was drained through a posterior mediastinotomy. Dilatations were re-established at weekly and bi-weekly intervals for the next three months. Reperforation of the esophagus in the lower cervical region occurred which was drained by a combined cervical and anterior mediastinal

approach. For the next two years regular weekly dilatations took place without marked improvement. The area of severest stenosis was in the lower cervical and upper mediastinal segments at the site of instrumental perforation. His nutrition was adequately maintained by gastrostomy feedings, but he was never able to take more than clear liquids by mouth. At times he was unable to swallow his saliva. He was presented to Thoracic Clinic for surgical consideration. After additional dilatations on the thoracic service, which were of no avail, it was decided to proceed with substernal jejunal transplant, using a procedure similar to the one employed in the second case.

There was abdominal exploration of the child and a segment of jejunum was mobilized. In the attempt to achieve sufficient length to span the entire esophagus the blood supply was impaired, and we were forced to resect the isolated jejunal segment. It then occurred to us that the right colon might serve as an esophageal substitute. Since the condition of the child was not too satisfactory at this time we decided to ligate the right colic artery in continuity and close the abdomen. Two weeks later the abdomen was re-entered after careful bowel preparation and the right colon was freed to the midtransverse colon. Caution was taken so that the midcolic artery was not injured. The ileum was then transected at the cecum and anastomosed to the distal transverse colon. The cecal end of the right colon was brought out through the neck after transplanting it substernally. The distal end of the colon graft was anastomosed to the upper part of the stomach. Three weeks later, after a rather uneventful convalescence, the esophagus was freed in the neck and transected. The distal strictured end was closed over and replaced. The proximal end was anastomosed to the colon behind the left sternocleidomastoid muscle. The colon was still quite edematous and bulky at this time, and, unfortunately, a stricture occurred at this site. A re-anastomosis was performed three and one-half months later when the colon had returned to normal size. The gastrostomy tube was removed and the opening closed spontaneously. At the present time the child has a well functioning alimentary tract. Eventually the distal esophageal segment may have to be removed, but there is no urgency. The child is eating normally for the first time in four years.

COMMENTS

Perhaps in time we will become more aggressive in dealing with such lesions, but so far we have been slow to advocate radical replacement if dilatations offer any chance of success. In children it would seem the right colon has certain advantages. From a technical point of view it is easier, quicker and, we believe, safer than jejunal transplants. Long term results may prove us to be in error, but to date we are extremely pleased with our results. We would strongly recommend that the procedure employed be done in two stages. In the case of colonic transplants it would seem well to wait several months before performing the cervical anastomosis.

Several problems remain unanswered. We are undecided regarding the desirability of whether or not to bypass the stomach. We have chosen to maintain normal gastrointestinal continuity in these infants. Their nutrition remains satisfactory. It would appear that there is no foolproof solution either to the problem of nutrition or recurrent peptic ulcerations. Our attempt to place the jejunum or colon as high in the stomach as possible will, we hope, reduce the incidence of regurgitant ulceration.

Theoretically it would seem desirable to excise the diseased or strictured esophagus. Practically, this has not proved necessary. So far a mucocele has not developed in either case; this parallels the experience of others. Certainly, we are mindful of this possibility, particularly in the second case. At this time surgical indications in the treatment of caustic strictures as well as congenital anomalies requiring reconstruction remain incompletely defined. This situation will continue until technics of esophageal reconstruction have demonstrated their reliability on a long term basis. Not until adequate follow-up observation has been made can we decide which of the many available procedures is superior. If a definite goal is realized perhaps earlier esophageal substitution will be justifiable and many a child will be spared the physical and psychic trauma of repeated esophageal dilatations.

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DISCUSSION

Ross Robertson (Vancouver, B. C., Canada): Reconstruction of the esophagus is a problem that has absorbed me for several years, so that I was pleased when Dr. Pinkham asked me to discuss his paper.

It has been an excellent and thoughtful presentation, and stimulates me to renewed efforts to assist these unfortunate children. The indications for reconstruction of the esophagus might well be extended and a more aggressive attitude adopted when one considers the trauma and complications of repeated dilatations. It also seems likely that more babies with tracheoesophageal fistula will be saved, as in Dr. Pinkham's case, by deferring reconstruction of the esophagus rather than attempting an insecure anastomosis under excess tension.

The anterior mediastinum is the best location for the reconstructed esophagus. It is remarkably easy to make the tunnel in this location, dissecting in normal tissue rather than in the posterior mediastinum scarred by previous inflammation. With reasonable care the tube of intestine can be threaded into the neck without damaging its blood supply. There is very little shock to the procedure and even small children stand it well.

Jejunum versus colon: It is true that in preparation of the tube of jejunum the blood supply may be found precarious and even insufficient in a few cases. On the other hand, the jejunum forms an excellent end-to-end anastomosis with the esophagus and, if isoperistaltic, functions most satisfactorily.

As Dr. Pinkham has stated, the jejunum is curved by its shorter vascular arcade and so tends to be redundant in the anterior mediastinum. This does not appear to distress the patient except for occasional audible peristalsis on swallowing.

Delaying the anastomosis in the neck is advisable if the viability of the transplant is doubtful. It is risky to postpone exteriorization of the upper segment of esophagus unless the child has learned to tolerate an excess of saliva in the mouth, as in Dr. Pinkham's third patient.

I have not tried using a tube of colon, but following this presentation I will be prepared for the opportunity. Dr. Robert Mustard of Toronto has used colon for reconstruction of the esophagus in adults. Because of its greater bulk he has preferred to split the sternum so that the tube of colon can be laid in the tunnel without endangering its blood supply.

The advisability of anastomosing the jejunum to the stomach is questionable because of the risk of peptic ulceration and stricture. It might be well to defer such an anastomosis until it becomes necessary to improve nutrition.

Removal of the residual thoracic esophagus has been unnecessary, the mucosa having been destroyed.

Since few of us have many of these problems to wrestle with, it will be a few years before the best solution is found. Dr. Betts of India may come up with the answer for us. He has an abundance of material and has already reported on several short term successes using jejunum in the anterior mediastinum.

In the meantime, it is wise when planning an esophageal reconstruction to prepare to use either jejunum or colon as determined by the vascular arcade.

Finally, Dr. Pinkham is to be congratulated on his excellent presentation and encouraging results.

Arthur J. Hunnicutt (Oakland, Calif.): The next time you perform a colectomy I believe that you will verify these findings: If you will expose the ileocolic vessels at their origin, the middle colic artery and vein, and the sigmoid artery and vein, you can clamp off any two of those and the blood supply will be intact to the colon for almost the entire distance from the cecum around to the sigmoid.

In turn, you can cut or clamp the left and right, or both left and right colic arteries and still not interfere with the blood supply.

In 1951 Dr. Samson and I had a case of lye stricture in an adult. We mobilized the colon from the cecum to the sigmoid. The only blood supply was from the middle colic artery. The cecum was anastomosed with ease, by Dr. Samson, to the esophagus above the arch of the aorta. The descending colon was put into the stomach.

The length of the ileocolic artery to the cecum, or the length of the middle colic artery to the transverse colon, will average about 6 inches.

The distance from that point to the esophageal hiatus will average 4 inches. Sometimes it will be 5 inches.

If you use only the middle colic artery for the blood supply, you can count on a viable right colon down to, but not always including, the cecum, and on a viable left colon down to the sigmoid.

Now, the length that you can pull up the intestine (the intestine itself is long enough to go above the neck) will depend entirely on the blood supply. The middle colic artery or the ileocolic artery will reach to the esophageal hiatus. From there on you have to depend on the arcs in the mesentery.

I think Dr. Pattison was one of the first to draw our attention to the fact that if you use the colon in this manner it makes no difference whether it is isoperistaltic or retroperistaltic.

Regarding the esophagointestinal anastomosis: In my last fifteen consecutive cases I have been using, rather than the usual two- or three-layer end-to-end anastomosis, the method of turning the esophageal mucosa back up on the esophageal wall as a cuff for about 1 cm. I put four stay sutures to hold it there, one in each quadrant; I then use these same sutures to bring the intestinal segment over the cuff. Thus the esophagus is telescoped within the lumen of the bowel.

Between each of these four stay sutures three to five additional sutures are required, so that the total anastomosis is made up of twenty-two, sometimes twenty-four interrupted sutures of No. oooo silk.

With the esophagus telescoped within the lumen of the anastomosed bowel, the swallowed foodstuff cannot hit the line of anastomosis and the circumferential scar is not within the lumen.

In these I have not hesitated to pass a tube down a few days after surgery, which most certainly I would not have done in an end-to-end anastomosis.

I think Dr. Pinkham's presentation has been very interesting. What segment of bowel, small bowel or large, will be best for esophageal replacement I do not know. The colon is easier to mobilize. The jejunum is cleaner and more readily available.

JOHN F. HIGGINSON (Portland, Ore.): I had not previously been aware of what might occasionally happen in strictures of the esophagus until last year, and I would like to discuss such a case with the type of reconstruction.

The patient was thirty-nine years old when I saw him last year. At the age of two and a half years he had swallowed lye and a stricture developed. He was started on the usual program of dilatations, perforations, drainages, and so forth.

Last year, early in the summer, a new type of dysphagia developed (he had also acquired a diaphragmatic hernia by shortening of the esophagus). Esophagoscopic biopsy showed that carcinoma had developed at the top of the esophagus.

A one-stage total esophagectomy was performed; and because it had to be total, I decided not to use the stomach, which had been our previous method of restoring continuity in esophageal resections. We therefore used the colon retrosternally. It was necessary to remove the right lobe of the thyroid to get it around, which is apparently not always necessary.

Both the preceding discussers and the essayists stated what fitted in with my previous understanding, namely, that removal of these strictured organs would not be necessary. However, at pathologic examination in my case the esophagus was a total mass of carcinoma from top to bottom, so that perhaps thought should be given in treating these children to what might eventually happen to some of these strictured esophagi.

WILLIAM P. LONGMIRE (Los Angeles, Calif.): I would like to thank Dr. Pinkham for a most interesting presentation.

In 1951, before this Society, we reported on two children upon whom esophageal reconstruction had been performed by antithoracic jejunal transplants. I have not heard recently of one of these cases, but I do hear from the other child at rather frequent intervals. He is now eleven years old and has developed and is progressing quite satisfactorily. He has an antithoracic reconstruction and there is considerable deformity over the anterior thorax. As a matter of fact, this child takes great delight in swallowing air when he is swimming and then floating around with this "inner tube" under the skin of his chest. His mother states very emphatically that the deformity has not influenced his personality development in any way.

In these two cases we learned quite definitely that in infancy it is essential that the lower end of the esophagus be anastomosed to the stomach.

When the stomach is bypassed in adult patients, they seem to get along quite well without food going through the stomach. However, both these infants lost weight and went downhill despite an adequate calorie intake until the jejunal limb was placed into the stomach.

If there is any place for the antithoracic type of reconstruction, it is in the congenital esophageal anomaly.

I am quite certain that it is hazardous to put the stomach or a coiled jejunal limb into the chest of a newborn child. I think one may exteriorize the

upper end of the esophagus and wait until the child is a year and a half or two years old and then use the substernal approach.

If it is desirable to complete the procedure before that time, however, it is probably necessary to put the jejunal limb outside the chest.

In our second case the entire procedure was completed by the time the child was six weeks of age. I think this has definite advantages.

PAUL C. SAMSON (Oakland, Calif.): I, too, have enjoyed Dr. Pinkham's presentation very much.

I would like to emphasize one point, if I may, and ask whether our experience is unique or whether others, particularly Dr. Pinkham, have had it.

Our experience was with two cases of lower esophageal stricture which were repaired by means of the ordinary pull-through of the stomach and resection of the stricture.

However, we have found in one of these children, as we have in several adults, that the patient continued to have symptoms of regurgitation and continued to have what seemed to be an enlarged stomach.

The end result of observation in one of these children and in several adults was that in many cases of esophageal resection we get a pyloric obstruction from what looks to be hyperplastic muscle and pyloric stenosis. In one of these children there was continued anemia because of gastric obstruction and distention with gastric bleeding, and eventually a pyloroplasty was performed.

This has impressed us so much that now in any case in which esophageal resection is performed (when necessarily you must perform a vagotomy) we have gone ahead and performed some type of pyloroplasty as a concomitant part of the operation. Perhaps that is one reason why we have recurrent esophageal regurgitations in some of these cases due to unrecognized gastric obstruction.

ROLAND D. PINKHAM (closing): I wish to thank the discussers.

I feel a little humble in reviewing these cases. Part of the point of presenting them was to gain additional information from the experience of others. This might be considered an additional reason for presenting papers at this meeting, aside from the indications Dr. Herrman mentioned.

The details of these procedures were included in the paper, but time did not permit a complete description. I am sorry that this was not possible, particularly in the third case, because it represented a series of interesting problems.

Regarding the telescoping of the esophagus, I remember years ago Dr. Eloesser advocated essentially that, and it is interesting to see it employed again. I have not tried it and would hesitate to do so for fear of creating a stricture.

As far as the remaining segment of esophagus (defunctionated) is concerned, there are two factors to be considered: (1) it should be done only in benign disease unless one is performing only a palliative procedure, and (2) removal should be carried out at a later date if indicated.

I think one reason we have had no trouble with peristaltic action in these two cases is that no vagotomy was performed. Our results in resection of the lower esophagus have been similar to Doctor Samson's. We were primarily interested in children in this report. I do not think there is any great difference between adults and children except, as Doctor Longmire brought out, the great need is for early nutritional care in infants.

The results of our two living cases are equally good. I would hesitate to say that the functional result of the large bowel substitution was better than the small bowel substitution. Certainly, technically it was easier to perform.

A Review of 155 Gastric Resections in Private Practice from 1946 to 1956

DAVID METHENY, M.D. AND VERNON O. LUNDMARK, M.D., *Seattle, Washington*

DURING the past ten years, from 1946 to 1956, we have performed 155 gastric resections in our private practice. (Table I.) There have been four operative deaths. Three deaths occurred after total gastric resections for carcinoma and in part were due to technical errors, as anastomotic leaks developed. The fourth death occurred after a subtotal resection in a woman with known cardiac disease, who had massive hemorrhage in a gastric ulcer. She was unconscious when first seen, but with adequate blood replacement she survived eight days. What we did not know at the time was that she had been on a 400 to 600 calorie diet for four months. Death was due to peritonitis. Postmortem examination revealed a leak at almost every suture. In all, there were 136 cases of peptic ulcer and the indications in this series are the recognized complications of benign ulcer. There were nineteen cases of cancer of the stomach; of these, five patients are living and well six months, four years, five and a half years, six years and ten years after surgery. Four of these survivors were thought to have had benign ulcer and so were early cases. (Table II.) Even though the possibility of curing early cancer of the stomach is given as a major reason for operating on so-called benign ulcers, we think that adequate surgery is the best treatment for benign ulcer as well, so we have been a little more liberal in our indications on operating upon benign gastric ulcer. Many of these cases are in the older age group and they constitute our best surgical results.

TABLE I

A. Data on 155 Resections

	1946–1951	1952–1956
Resection for carcinoma (19)	11	8 (5 survivals; 6 mo.–10 yr.)
Resection for benign ulcer (136)	71	65
Billroth I	7	49
Billroth II	70	23
Postoperative complications	26 (13 patients)	10 (5 patients)
Postoperative deaths	3 (1 benign)	1

B. Recurrences of Ulcer (7)

Procedure	Duodenal Ulcer	Gastric Ulcer	Marginal Ulcer	Reoperation
Billroth II	2	..	1	1
Billroth I	3	1	..	2

TABLE II

TYPE OF CASE

Disease	1946–1951	1952–1956	Total 1946–1956
Duodenal ulcer	40	44	84
Gastric ulcer	25	19	44
Duodenal and gastric ulcer	2	2	4
Marginal ulcer	4	..	4
Total benign	71	65	136
Carcinoma	11	8	19
Total	..	..	155

COMPLICATIONS

We had twenty-seven significant complications in thirteen cases in the series from 1946 to 1951 when the Billroth II operation predominated; we had ten postoperative complications in five cases in the series from 1952 to 1956, during which time the Billroth I operation was the predominant technical procedure. Table III shows the complications as they occurred in the entire series for these two periods. It will be

noted that in the second series the greater number of complications occurred in the Billroth II procedures in both series. The change to the Billroth I procedure was initiated to a large extent by our dissatisfaction with the immediate postoperative complications that we had encountered and the technical hazards that are inherent in the Billroth II procedure, namely, the hazard of the duodenal stump, proximal loop stasis and kinking of the anastomosis or efferent limb. In view of our experience with the Billroth II group, we began a second series in which we used the Billroth I operation wherever feasible. All our patients with cancer and 124 of the 136 patients with peptic ulcer have been followed up to within the last six months. Table IV shows the breakdown as to the type of surgery performed in the two series. In the first five years there were seven Billroth I and seventy Billroth II operations and five total gastrectomies for carcinoma. In the last series forty-nine Billroth I and twenty-three Billroth II operations and one total gastrectomy for carcinoma are shown. It will be noted that a certain small group of Billroth I and Billroth II operations were performed for carcinoma and they are included in the aforementioned series. All in all, these account for 155 consecutive gastrectomies performed for benign and malignant disease.

TABLE III
POSTOPERATIVE COMPLICATIONS IN 155 CONSECUTIVE GASTRIC RESECTIONS FOR BENIGN ULCER AND CARCINOMA

	1946–1951	1952–1956
Atelectasis and pneumonitis	1	2*
Pulmonary infarct	1	..
Phlebitis	1	1*
Wound dehiscence	3	..
Pancreatitis, acute	2	1†
Pancreatic edema	3	1*
Pancreatic fistula	1	..
Subphrenic abscess	1	1†
Subhepatic abscess	1	1†
Anastomotic leak	3	..
Wound infection	2	..
Stomal obstruction	2	1†
Postoperative bleeding	1	..
Subdural hematoma	..	1†
Ruptured diverticulum of colon (peritonitis)	..	1*
Massive hemorrhage and psychosis	1	..
Proctitis, antibiotic	1	..
Intestinal obstruction, high	2	..
Total	26 (13 patients)	10 (5 patients)

* Billroth I procedure.
† Billroth II procedure.

In the first five years, as well as we can estimate, the average blood loss during surgery was 460 cc. and the average postoperative hospital stay was 12.8 days. In the second series the average blood loss (measured) was 430 cc. and postoperative stay was 7.4 days. In the second series not only are there fewer complications but also they were much less serious. We think this is related to the increased use of the Billroth I type of operation. It is significant, too, that in the second series the most serious complications were in the Billroth II procedure.

TABLE IV
TYPE OF PROCEDURE

Procedure	1946–1951	1952–1956	Total 1946–1956
Billroth I	7	49	56
Billroth II	70	23	93
Total gastrectomy for carcinoma	5	1	6
Total	..	..	155
Billroth I for carcinoma	4	4	8
Billroth II for carcinoma	2	3	5

TABLE V
BLOOD REPLACEMENT AND LOSS IN GASTRECTOMIES
(Average, All Cases)

Data	Blood (cc.)
Blood preoperatively	1,080 } 1,730 used or replaced
Blood during surgery and postoperatively	650 }
Blood loss, Billroth I and II	450
Mean loss, Billroth I (last 20 cases)	360

In the last twenty Billroth I cases the mean blood loss was 360 cc. measured. In the matter of preparation of these patients we have used 1,080 cc. of blood on the average per case preoperatively; during surgery and the postoperative period we have used 650 cc. or a total (use and replacement) of 1,730 cc. In both procedures the average blood loss has been about 450 cc., as previously noted. (Table v.)

We think the improved results in our second series are due to several factors. First, the

TABLE VI
1946–1951 GROUP

Disease	Operative Deaths	Since Died	Lost to Follow-up	Success	Improved	Failure
Duodenal ulcer (40)	..	2	4	20	12	2
Gastric ulcer (25)	..	2	5	15	3	..
Duodenal and gastric ulcer (2)	..	..	..	1	1	..
Marginal ulcer (4)	..	..	..	1	2	1
Total benign	..	..	..	37	18	3
Carcinoma (11)	3	7	..	1	..	..

TABLE VII
1952–1956 GROUP*

Disease	Operative Deaths	Since Died	Lost to Follow-up	Success	Improved	Failure
Duodenal ulcer (44)	..	..	2	26	13	3
Gastric ulcer (19)	1	2	..	13	2	1
Duodenal and gastric ulcer (2)	..	..	1	1	..	..
Total benign	..	..	..	40	15	4
Carcinoma (8)	..	3	..	5 living†	..	..

* There were no marginal ulcers in this group.
† Patients living 6 mo., 4 yr., 5½ yr., 6 yr. and 10 yr.

Billroth 1 operation is confined to the upper abdominal compartment and is technically easier and faster. Second, the use of Banzet clamps we believe does for gastric surgery what the Pott's clamp has done for arterial surgery. It gives mechanical and hydrostatic control of the stomach easily and without crushing injury. It allows for adequate high resection and open abuting anastomosis without inversion and stenosis. It has completely prevented the stomal problem in our second series of cases. We have had no postoperative bleeding. Third, we have learned to better prevent homeostatic impairments; our preoperative blood volumes are better adjusted, and it is our belief that operative blood loss is better replaced cubic centimeter for cubic centimeter as it is lost. Last but not least, postoperative gastric suction has been used less and less. In the last series we have used it only two times, once in an elderly man in whom pancreatitis and jejunal obstruction developed postoperatively, and once in an elderly man in whom a ruptured diverticulum of the sigmoid and peritonitis developed on the fourth postoperative day. Therefore the electrolyte problem has hardly existed. As a result, there were no significant complications from nasal tubes, and patients slept better and required less sedation. Postoperatively we have attempted to approximate intravenously the normal fluid and electrolyte loss that these patients will average until peristaltic activity commences. We average 2,000 cc. of 5 per cent glucose with 28 mEq. KCl, and 500 cc. of Darrow's solution in twenty-four hours. This gives 60 mEq. of sodium and about 45 mEq. of potassium. Fluids by mouth are not given until flatus is passed by rectum spontaneously. Then liquids without carbohydrate are given 1 ounce hourly and increased as tolerated. This usually starts forty-eight to seventy-two hours postoperatively.

Should retching or vomiting occur after surgery, it is a dramatic event. The treatment is not so much gastric suction as diagnosis. In our experience these cases have been due to such

TABLE VIII

Case	Type of Ulcer Treated	Operation	Type of Recurrence	Treatment after Recurrence
I, E. St. C. ♀	Duodenal	Billroth I → Billroth II and gallbladder	Stomal	Medical; good response
II, J. B. ♀	Marginal	Gastroenterostomy → Billroth II	Stomal	Massive hemorrhage, death (psychosis)
III, C. P. ♂	Duodenal	Billroth II	Stoma	Medical; good response
IV, G. K. ♂	Duodenal	Billroth I	Duodenal	Medical; good response
V, C. B. ♀	Duodenal	Billroth I → Billroth II	Duodenal	Good result (3 recurrences in family)
VI, M. A. ♀	Duodenal	Billroth I	Duodenal	Medical; slow healing, multiple allergies
VII, M. B. ♂	Gastric and neurofibroma	Billroth I	Duodenal	Medical; good response

minor causes as morphine sensitivity or something not related to the stomach. We had vomiting from a localized peritonitis of a ruptured sigmoid diverticulum, a subdural hematoma and acute pancreatitis, to note the three major complications of our last seventy-three cases.

A finding that seemed interesting to us was the significant number of our patients with peptic ulcer who also had gallbladder disease of such magnitude as to require gallbladder surgery either before, during or after gastric resection. We have twenty-six such cases in our series, or approximately one case in six. In our most recent case stones developed two years after gastric resection. Her gallbladder was completely free of stones at that time. Quite a few of our cases of postoperative discomfort have resulted from gallstones which developed after gastric resection.

RESULTS

All cases in which postoperative ulcers developed demonstrable by x-ray were considered failures no matter how slight the symptoms might be. In the improved group were those patients who had definitely derived benefit and were working full time but occasionally had some symptoms referable to their digestion. Those patients placed in a satisfactory or excellent category were those having no digestive symptoms and who had regained their normal preoperative weight or better. Tables VI and VII show the breakdown of our two series. In the first series, which were predominantly Billroth II procedures, we had three failures in Billroth II procedures. Of the remainder of the fifty-eight cases, thirty-seven were considered excellent and eighteen were considered improved. In the second series there were four failures, forty were deemed successful and fifteen were improved. In our cases of gastric ulcer (thirty-four cases followed up) there were no failures in the first series and one failure in the second series in a Billroth I procedure. This man had a superficial ulceration that healed rapidly with medical management. He was a person who devised his own postoperative rules which were quite bizarre.

Our experience with the duodenal ulcers shows the problem is a bit more complex. There were three recurrent duodenal ulcers in the second series, all in Billroth I operations. There were two recurrences of duodenal ulcer and one of marginal ulcer in the first series, all in Billroth II operations. The stomal recurrence was a second recurrence following a Billroth II procedure in a patient with acromegaly who eventually died in a mental institution with recurrent stomal ulcer and massive hemorrhage.

Recurrences in Benign Ulcer

Table VIII shows the breakdown of the cases of recurrent ulcer. One recurrence was in a fifty year old secretary who had considerable postoperative distress and x-rays eventually showed a duodenal ulcer. She had undergone a Billroth I procedure. Her response to medical management was sluggish. Case II is the only other real problem we had in this group. The remainder have healed rapidly with conservative medical management. X-rays have shown shallow ulcerations. We are of the opinion that no definite conclusions can be drawn in our

Billroth I group with duodenal ulcers for another five to ten years. The recurrences may be even higher than we expect. It will be noted that three revisional operations were performed for the recurrences. In Case I secondary resection was performed with removal of the gallbladder for stone with a stomal recurrence. The second case was the patient with acromegaly who underwent gastroenterostomy elsewhere and a revision to a Billroth II operation. Subsequent stomal ulceration resulted in massive hemorrhage and death while the patient was in a mental hospital. The third revision occurred in a woman who underwent a Billroth I operation. A duodenal ulcer developed and the procedure was subsequently revised to a Billroth II because we believed there had been excessive tension on the original gastroduodenal segment. Although we have come to no definite conclusions in the Billroth I group, this much may be said however, for our cases: If patients have trouble postoperatively, it is not from stomal edema, proximal loop stasis, loop kinking, duodenal stump leak or postoperative bleeding; it is usually related to recurrent ulceration and usually responds well to medical management. Two of the three revisions are now in the successful category. We have not supplemented any of our cases with vagotomy to date.

POSTOPERATIVE CARE

Postoperative distress, and fullness after meals, was rather common occurring in the early postoperative months. It usually improved with time, usually within six months. When it persisted, it was never severe enough to cause economic crippling. Our improved group occasionally had some symptoms suggesting hypoglycemic manifestations, but with proper instruction these have practically disappeared. Postoperative distress has come from other diseases such as gallstones, as mentioned before, and one case of cancer of the pancreas. In one case acute pancreatitis developed four years after gastric resection. One patient had a subdural hematoma resulting from a head injury sustained when he fainted from blood loss prior to resection. Postprandial distress or "dumping" seemed, in our cases, directly related to the amount of stomach removed and the tension on the new gastroduodenal or gastrojejunal segment. We believe that in some of our early cases there was some tension on the reconstructed segment, especially in Cases V and VI in our recurrences, which was probably due to technical error. Since that time a revision of our technic has improved this problem, we believe. Much of this distress, minor as it has been, departs with time.

We give the following instructions to our patients:

1. Take small, frequent feedings, preferably six, until the upper gastrointestinal tract has become accustomed to the new situation.
2. Excessive fatty foods and sweets in the form of syrups, candies, etc., should be restricted. Many people cannot take whole milk for variable periods of time after gastric resection.
3. Liquid should be eliminated from meals and should best be taken between feedings.
4. Ice cold drinks in large amounts should be avoided.
5. Tobacco and alcohol are discouraged.

TECHNIC

The technic we employ today in the majority of our gastric resections will be illustrated in Figures 1 to 3. The abdomen is entered through a generous midline incision. After careful exploration the greater curvature is freed up, ligating the gastroepiploic vessels and removing the omentum to the first vasa brevia vessels proximally and to the pyloric ring distally. The lesser curvature is freed up in a similar manner, doubly ligating the left gastric vessels near the take-off at the splenic artery. The duodenum is freed up laterally by blunt and sharp dissection, and the first and second part of the duodenum is reflected medially by the Kocher maneuver so that the inferior vena cava is exposed in the process. This may be easily done in the gastric ulcer and occasionally in carcinoma of the stomach; but in some duodenal ulcers, especially where scarring and foreshortening are present, or where a low-lying ulcer penetrating the pancreas is present, it may be extremely difficult. The mobilization can be simple or extremely difficult. We make a practice of not freeing much over 2 plus cm. below the pyloric ring on the posterior and superior surfaces of the first part of the duodenum, so as not to impair the blood supply to the duodenum and otherwise injure the accessory pancreatic duct. (Fig. 1.) At this point no comprising steps have been taken. If enough mobility is obtained and a gastroduodenal

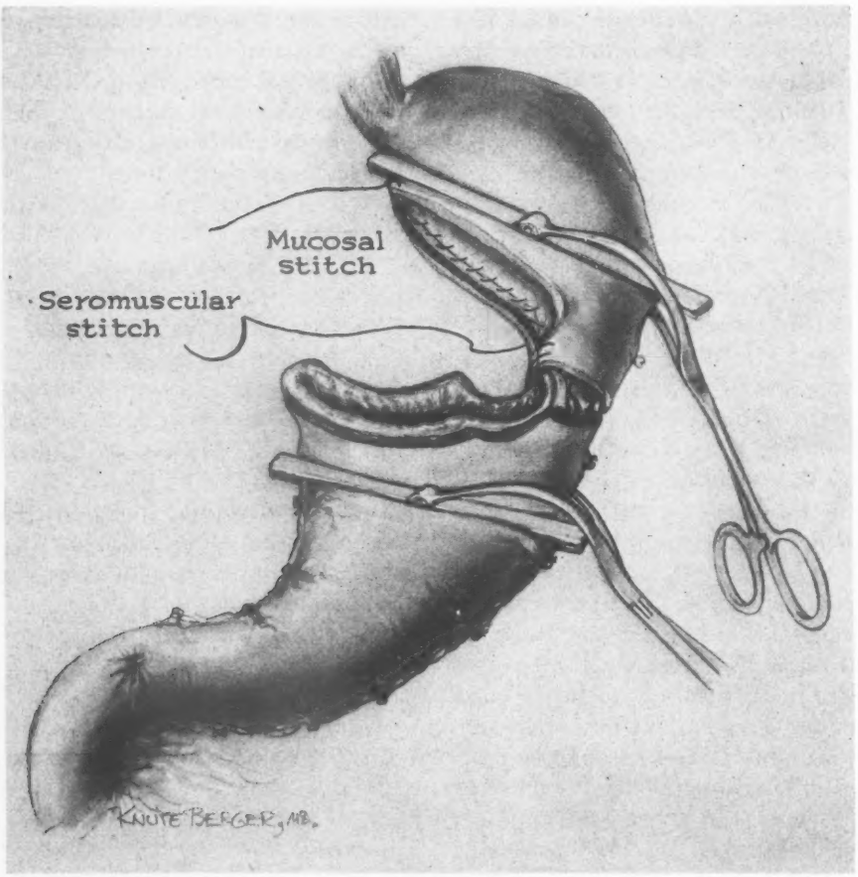

FIG. 1.

anastomosis is elected, the Banzet clamp is applied well above the elected site of proximal section, as shown in Figure 2. The seromuscular layers are incised down to the mucosa on both anterior and posterior surfaces of the stomach and at the pyloric ring or just below the duodenal ulcer. If an additional 1 to 2 cm. is needed for better mobility of the segment, a short tunnel is created on the greater curvature side of the stomach. In the Billroth II operation this is not necessary. The mucosa is sectioned at the duodenum, and the duodenal secretions are suctioned off and a dry sponge is inserted into the lumen to prevent spillage. The first suture is placed on the lesser curvature (Fig. 2) using No. 00 chromic catgut with an atraumatic® needle through the seromuscular layer, and the mucosa is then sectioned down to the tunnel on the greater curvature side. The mucosa is sutured with a loose running stitch and then the return suture approximates the seromuscular layer. The lock stitch is frequently used in this layer. This abuting type of anastomosis can then be reinforced with a series of interrupted No. 000 silk sutures. The Banzet clamp gives excellent control of the upper segment and controls bleeding. The gastroduodenal anastomosis is then performed using a soft enterostomy clamp above the tunnel, primarily to control bleeding. The posterior row of No. 000 silk is laid through the seromuscular layers of the tunnel and the duodenum, as shown in Figure 3. The mucosa is then sectioned and closed with a running No. 00 chromic catgut using a running or lock-type suture posteriorly and then anteriorly. The anterior seromuscular layer is then closed with another series of interrupted silk sutures. The final reconstructed segment should hang loosely, and if properly mobilized will do so. Mobilization of the duodenum using the Kocher maneuver, the formation of a short tunnel and the abutment type of open anastomosis, all contribute to an easy-lying anastomosis that allows a high and adequate resection free of tension at the suture line. The gastroduodenal anastomosis

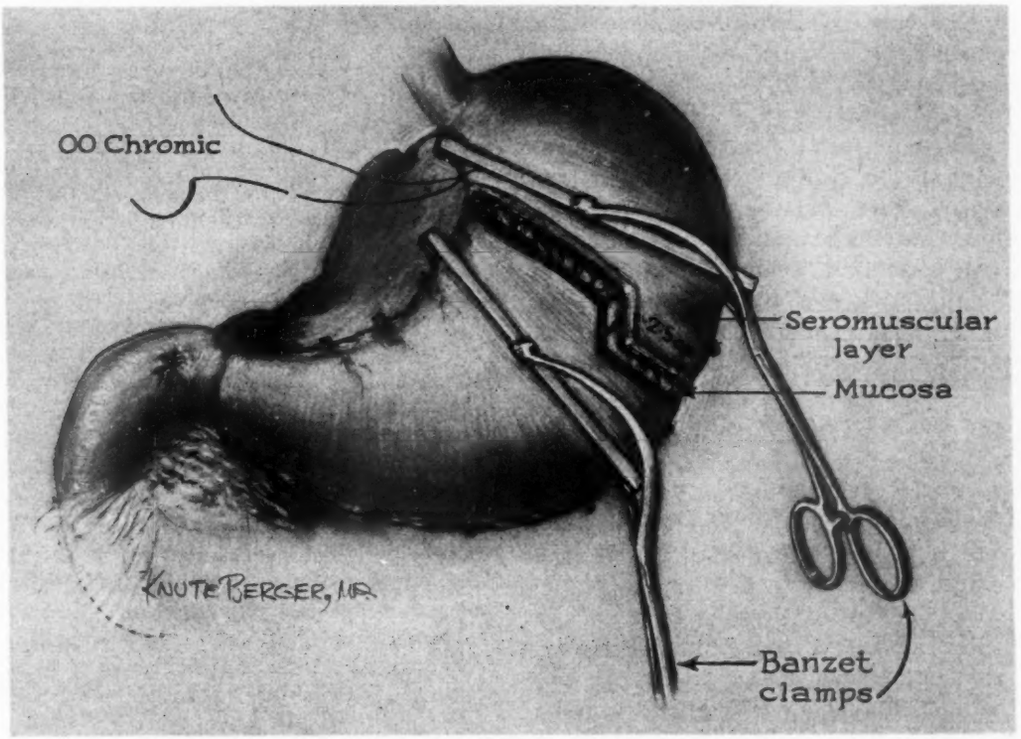
00 Chromic
Seromuscular layer
Mucosa
KNUTE BERGER, M.D.
Banzet clamps

FIG. 2.

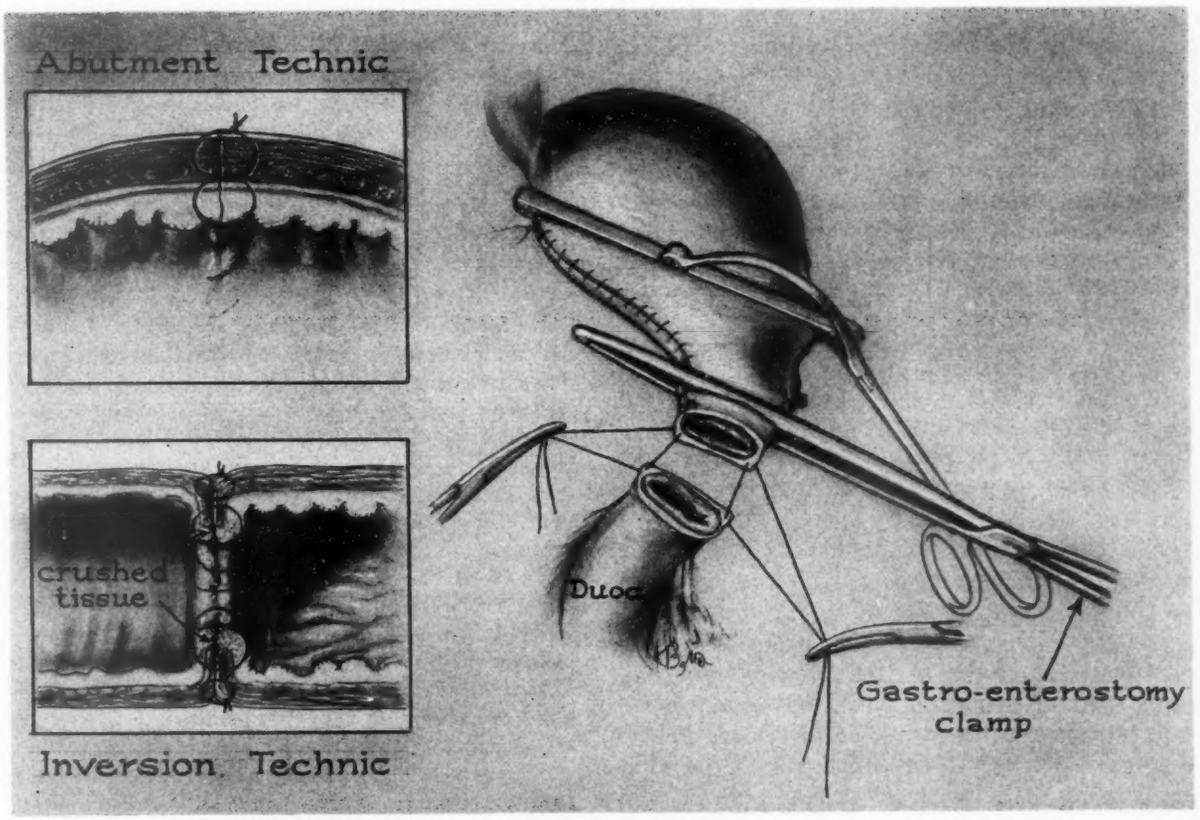
Abutment Technic
crushed tissue
Inversion Technic
Duod.
Gastro-enterostomy clamp

FIG. 3.

contains no crushed tissue to encourage edema; end-on abutment allows greater mobility and discourages inversion and stenosis. The best proof of the latter fact is that we have had no stomal problem in our last series of seventy-three cases. If the Billroth II operation was used because a high resection might have been compromised for lack of mobility or from whatever cause, preliminary mobilization aided closure of the stump of the duodenum; and regardless of whether an antecolic or retrocolic, isoperistaltic or antiperistaltic repair was used, the stoma was a better one using the abutment technic and Banzet clamp.

SUMMARY

We believe that further time will be necessary to evaluate our long range results with the Billroth I operation. The technic that we have outlined presents the advantages that can be used in either the Billroth I or Billroth II operation. Early complications are minimal in the Billroth I operation using the Banzet clamps and the end-on abutment reconstruction which we have described. It favors a mobile resected segment free of leaks and stenosis. If postoperative distress develops, it is usually due to recurrent ulceration in the Billroth I procedure and the signs are definite. We believe it is the operation of choice in the gastric ulcer and early gastric carcinoma. In our hands the operation is technically a simpler procedure, less time-consuming, and the resection is adequate. Late distress may be related to cholecystitis, pancreatitis and other factors that should be searched for.

ADDENDUM

Since this paper was presented, we have had three additional recurrent ulcers, one after the Billroth II procedure and two after the Billroth I. This increases the recurrences to ten recurrent ulcers proved by x-ray. Although the symptoms of these recurrent ulcers are not severe, the increasing number of recurrences as time goes on raises a philosophic question: Twenty-five years from now will not classic gastric resection be in the same category as gastroenterostomy is today? There is a definite question in our minds whether accurate and careful follow-up will not indicate a comparable recurrence rate in other centers about the country.

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DISCUSSION

Wm. B. Hutchinson (Seattle, Wash.): The evaluation and comparison of gastric procedures on any group or groups of patients with peptic ulceration is incredibly difficult. This, in my opinion, is due to the great variability of the most important single factor, that of selection of the candidate for surgery. I am positive that if our criteria of operability were as rigidly drawn on our patients as they would be by the various members of this audience if they were to submit to surgery for peptic ulceration, most of our postoperative problems would cease to exist. Whether a Billroth I or II was necessary and whether it was performed retrocolic or antecolic 6 or 12 inches from the ligament of Treitz would seem trivial. The patient with really severe long-standing ulcer pain will be grateful for relief. This is not the person who complains about numerous minor postoperative problems. The patient with functional complaints mixed up in his story which is of short duration makes one wonder if he is the best possible candidate for surgery. This patient will most frequently have a disappointing result. The disappointment is too often attributed to the type of resection performed. Repeat operations often fail because he was not really a suitable candidate for surgery in the first place.

The authors, in presenting their ten-year experience in private practice with gastrectomy, have given us a more personal follow-up study than we would have reason to expect from a larger institutional survey. Their mortality rate of one death in 136 resections for benign peptic ulceration is excellent. Their survival time in four of the nineteen cases of carcinoma of the stomach is also excellent. In spite of the fact these patients were operated upon for benign ulceration and supposedly "gotten early," I am sure the essayists would not mean to convey the impression that the size of ulceration or tumor mass and curability were synonymous. We should not forget that the Billroth I in selected cases is a good operation for cancer as well as ulcer.

The feasibility of using a Billroth I procedure revolves around the local situation in the duodenum in each given case. In the duodenal ulcer of long

standing, the one that probably satisfies the criteria for operability best will often be a hazardous one for which to perform a Billroth I procedure for reasons of deformity, scarring and inflammation. Any time too many tricks have to be resorted to in order to use the Billroth I procedure the issue is being forced and probably the Billroth II is more desirable. As time has gone on, the authors have unquestionably learned more about the technic of gastric surgery, and the Billroth I procedure in particular, so that must be taken into consideration when they compare the results of their two series.

The Banzet clamp is a hemostatic, non-crushing clamp which with the tunnel cuff has combined in this series to give a satisfactory resection. However, with the high incidence of stomal ulceration reported one must ask if the authors were forced to reduce the extent of resection because of the Banzet clamp. Generally speaking, whatever other defects are associated with high resection for peptic ulceration, recurrent marginal ulcer is not one of them.

As a result of Dr. Henry Harkins's repopularization of the Billroth I procedure we, like the authors, have used this operation with progressive frequency in the past five years. We have been uniformly happy with our results.

It has been a privilege to discuss this paper.

RALPH H. LOE (Seattle, Wash.): It is always worthwhile to hear from the personal experience of accomplished surgeons. I believe this gives a different evaluation than, for instance, a collection of cases taken from many services or from one charity institution. Certainly the results will not be the same. The mortality rate of 0.6 per cent for subtotal gastrectomy is very commendable. I am quite sure from the relatively small number of totals reported by Drs. Metheny and Lundmark that this procedure is performed only when absolutely necessary to remove the growth. I am quite in favor of the radical subtotal for carcinoma of the stomach unless the operation is compromised by the extent of the growth. The morbidity rate is much smaller.

By leaving a small nubbin of stomach one prevents reflux of material into the esophagus by preserving the cardioesophageal valve, a much more substantial anastomosis is permissible and sufficient gastric mucosa is preserved to prevent in most instances development of severe anemias.

I would certainly agree with Dr. Lundmark that the Billroth I operation is easier and faster than the Billroth II. I like it particularly for gastric ulcers and I have used it also for carcinoma of the stomach, but I am not at all sure that it is as good an operation as the Billroth II for this purpose. We cure only a relatively small percentage of gastric carcinomas, so here palliation is important. I now have a patient in the hospital in whom an obstruction has developed from recurrent carcinoma around the pancreas following the Billroth I operation. If I had performed the Billroth II, I think his palliation would have been better.

It would appear to me that Dr. Lundmark's recurrence rate, while it is relatively low, is about the same for the Billroth I procedure as the Billroth II. However, if that rate can be maintained over the years, the pendulum would still be in favor of the Billroth I because of the other advantages which have been enumerated. Of course this is the crux of the situation, and as far as I am concerned time alone will settle the problem. Several authors have now reported a much higher recurrence with the Billroth I procedure; however, in these cases it is quite probable that very few underwent a ¾ gastric resection as advocated by Harkins and Moore. Walters admits, for instance, that in the relatively few cases that have been followed up by them or others with a ¾ resection there has been a rather low recurrence rate.

In one of my Billroth I procedures, which I performed for a penetrating marginal ulcer following a Billroth II, a suture line leak developed and it was interesting to see that no bile was coming out of the sinus and there was no digestion of skin. This may be significant in the neutralizing effect of pancreatic and bile juices on gastric content. Should one, then, perform an end-to-side Billroth I procedure to bring the stomach closer to the ampulla of Vater, as Dr. Hinshaw showed us? For this reason I have favored a Finney pyloroplasty over a Heineke-Mikulicz procedure when performing a vagotomy.

I was interested to see the high rate of gallstones associated with peptic ulcer following gastric resection. I have also seen the reverse in several instances, i.e., ulcers developing after cholecystectomy. What the relationship is I do not know, but I suspect there is more to the control of duodenal ulcers in the duodenum than is known today. Most of our surgery of today is directed to the control of acid pepsin from the stomach side.

I would gather from Drs. Lundmark and Metheny's remarks that they have not been intrigued by vagotomy or other operative procedures for duodenal ulcer. My own reaction is to try to tailor the known effective procedures to fit individual cases. How successfully one can do this, of course, remains to be seen. There are certainly many anatomic and physical facts which can make one lean toward one operative procedure or another, such as underweight, overweight, emotional and mental status, degree of acidity, drinking habits and anatomic findings at the time of surgery.

I shall watch with interest Drs. Lundmark and Metheny's continued long term follow-up on their Billroth I cases for duodenal ulcers. It has been a pleasure to discuss the paper.

GORDON K. SMITH (Los Angeles, Calif.): The

problem of surgery of peptic ulcer of the duodenum should not be included in the discussion of carcinoma and gastric ulcer. They are entirely different diseases and need completely different operative procedures.

I wish to emphasize one or two points. The first is that no one type of surgical procedure will fit all patients and, secondly, that no surgical meeting is complete unless there are at least two papers on peptic ulcer. Quite obviously, if the completely satisfactory operation for duodenal ulcer had been developed, we would not keep talking about it at every meeting.

The first essential of any surgical procedure for peptic ulcer should be a low morbidity and mortality rate. We seldom transfuse any more during operative procedures for duodenal ulcer except when the patient has had preoperative hemorrhage or is bleeding at the time of surgery. The operative procedure should have a reasonable assurance for a good long term result.

Other authors, as has been pointed out by the discussers, have reported as high an instance of recurrence following the Billroth 1 operation as 28 per cent. Dr. Farris and I recently made a detailed questionnaire of 60 of 100 patients on whom vagotomy and some type of complementary procedure were performed. There were no recurrent ulcers, no deaths and no serious complications. Ninety-three per cent of these patients had satisfactory results and were restored to good economic status.

Dr. Joseph Weinberg of this society has done pioneer work in popularizing the operation of vagus nerve section. At the present time I believe he has the largest number of patients on record who have had vagotomy.

We believe, as does Dr. Weinberg, that the causes of failure of vagotomy are, first, incomplete vagotomy and, secondly, an inadequate complementary procedure. At the present time our preference is for vagotomy and pyloroplasty when feasible. If technical difficulties arise, we perform either a gastroenterostomy or a low hemigastrectomy.

Many people who advocate gastrectomy of two-thirds to three-quarters of the stomach use vagotomy for recurrent marginal ulceration. We believe this thinking to be in reverse. Why not perform the conservative procedure first, and then in those few cases in which the operation fails perform a gastrectomy? Geography seems to play an important part in the type of operation performed for duodenal ulcers on the Pacific coast. Very few people outside the city limits of Los Angeles, which extends almost to the Tehachapi mountains, believe that vagotomy is of any value at all. It is our firm belief that vagotomy should be part of the armamentarium of every surgeon. It should not be performed necessarily in every case, but it is a less formidable procedure than gastrectomy and many seriously ill patients may survive vagotomy where gastrectomy with excision of the ulcer may lead to tragedy. It is also our duty in training residents to see that they are familiar with more than one type of procedure.

I have enjoyed Drs. Lundmark and Metheny's paper. They are to be congratulated on the low mortality they have achieved in the Billroth 1 type of operation.

WILLIAM P. LONGMIRE (Los Angeles, Calif.): I would like to thank the authors for presenting the information outlined in this paper. I think one of the great points of significance here is that it represents the personal experience of two members of our society.

From a physiologic standpoint, the duodenum is a most interesting endocrine organ. However, to me it has been very disappointing that we do not have better evidence of improved functioning following gastric resection when alimentary continuity through the duodenum is restored, such as is the case in the Billroth 1 operation.

After a rather limited experience, extending over a period of several years we have about come to the conclusion that the discussion of the Billroth 1 vs. Billroth 11 operation is generally taking its place beside the discussions of silk vs. catgut and anterior vs. posterior gastroenterostomy in surgical conversations when the evening is late and the conversation is lagging.

I would like to call to the attention of the members of this society a monograph published in 1954 by Wallenstein.

Wallenstein: Postcibal symptoms
Entire series

None	53.9%
Occasional	20.6%
Mild	13.1%
Severe or moderate	12.4%

Grade	*Billroth I*	*Billroth II*
None	54.2%	53.8%
Occasional	26.8%	16.9%
Mild	13.7%	12.8%
Moderate or severe	5.3%	16.6%

This monograph fails to have the personal touch of the series reported here, but it presents Wallenstein's series of 1,256 operative cases followed up from three to twenty years.

Here we see in his series of cases the postcibal symptoms in the Billroth 1 and the Billroth 11 procedures. Actually the series in which there were none are about the same in the two groups; the only difference of significance is in the severe symptoms where the patients undergoing the Billroth 11 procedure had a rather marked increase over those undergoing the Billroth 1.

Without Recurrence

Method	*10 yr.*	*20 yr.*
Billroth I	92%	87%
Billroth II	97%	92%

Mortality

Billroth I	1.9%
Billroth II	4.4%

Difference not statistically significant

As far as postoperative weight gain is concerned, in the Billroth I procedure there seems to be some better weight gain than in the Billroth II throughout the series.

In the presence of anemia this is broken down between men and women. The presence of anemia was greater in the group following the Billroth II operation, particularly in the women.

Wallenstein: 1,256 cases

	Postoperative Weight		
Method	*+5 kg.*	*0*	*−5 kg.*
Billroth I	57%	38%	5%
Billroth II	37%	50%	13%

Anemia*

Method	*Men*	*Women*
Billroth I	59%	28.3%
Billroth II	74%	61%

* < 13.6 gm. % hemoglobin—♂
< 11.5 gm. % hemoglobin—♀

When we come to discuss the problem of recurrence at ten years (and I think this gives us some of the statistics which many of us would like to know because these are patients we followed up ten and twenty years) the rate of the Billroth I without recurrence is 92 per cent, and the Billroth II 97 per cent.

At the end of twenty years the rate of the Billroth I without recurrence had dropped down a little further and that of the Billroth II was still a little bit better.

The mortality rate in this series of Billroth II anastomosis was a little greater but not of statistical significance, the author states.

Wallenstein concluded by recommending the Billroth II resection in the case of men with duodenal ulcer, and the Billroth I operation for men with gastric ulcer and women with either gastric or duodenal ulcer.

It does seem to me that this is "slicing the cake right thin," and probably points out again that the difference between the two procedures is of not great significance.

VERNON O. LUNDMARK (closing): I want to thank the discussers for their comments, and I certainly wish to thank Dr. Longmire for the added data that he supplied in the larger series of cases.

I would like to say a word with regard to the adequacy of resection. This had been pointed out as a criticism of the Billroth I. I think that with the particular clamp which we employ we are able to control the proximal segment much more easily, and I believe that we have been performing 70 to 75 per cent resections. As a matter of fact, I think that some of the problems we have had postoperatively have been storage problems, and I think that certainly indicates one can perform a perfectly adequate or high resection.

As far as the problem of vagotomy is concerned, we have had but little experience since our early cases were uncomfortable experiences. When I started to perform resections that seemed to have a smoother postoperative course, I stayed with the technic that worked well for me. To date this happens to be the procedure that works well for me and it is not one where I restrict it entirely to the Billroth I or Billroth II, as our data certainly indicate. In the second series of our cases the Billroth I procedures were performed twice as frequently as the Billroth II procedures. However, we do not try to perform the Billroth I where it is not feasible. If after mobilization of the stomach and the duodenum an easy-lying anastomosis with a high resection can be performed, we use the Billroth I operation as the procedure of choice.

Management of Massive Hemorrhage from Peptic Ulcer

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THERE is no more difficult judgment than that required of the clinician when he must evaluate the inherent lethality of an acute, severe, hemorrhage from a peptic ulcer. For adequate orientation regarding the problem he must be provided with careful analyses of significant numbers of such situations. These studies should include all patients with acutely bleeding peptic ulcer who enter the hospital, whether or not they are operated upon. Further, rigid criteria of massive hemorrhage must be applied in order to exclude all instances of lesser degrees of bleeding. Additionally, because of the extreme influence of the patient's age and the presence of serious associated disease on this outcome, these factors must be clearly presented.

In order to appraise the validity of existing indications for surgery it is admittedly desirable to have the most exact data obtainable regarding a given patient. However, since many of the precise technics are not available in the average hospital a study using generally available methods should have special value. It is our purpose to report such an analysis of consecutive cases of massive hemorrhage from gastric or duodenal ulcer, including all patients admitted to the University of Southern California Medical and Surgical Services at the Los Angeles County Hospital over a period of one year from July, 1954 to July, 1955.

Another phase of this study of massive hemorrhage from peptic ulcer has to do with estimating the present benefits of operative therapy based on utilization of the knowledge gained from the reports of studies such as those of Stewart [1], Welch [3], Dunphy [6], and Mathewson [2]. We have made such an analysis of our cases in order to report results presently being obtained, and have attempted to identify potential means of improving our future results.

CRITERIA OF MASSIVE HEMORRHAGE

In order to define massive hemorrhage it is necessary to establish criteria that eliminate all instances of acute bleeding that were of moderate degree and would have recovered spontaneously, or with relatively conservative therapeutic efforts. Only when such cases are excluded can the effectiveness of therapy be measured. No cases are included in our series unless they were of such severity as to fulfill the following criteria.

Acute hemorrhage accompanied by either:

1. Acute hypovolemic shock
   Systolic blood pressure of 90 mm. Hg or less
   Cold skin
   Sweating
   Pallor, and/or

2. A hemoglobin of 7.5 gm. or less per 100 cc.

Sixty-three of the seventy-three patients in this series had acute hypovolemia productive of the signs of shock. The circulatory disturbance of the remaining ten patients had stabilized when admitted, but the bleeding was severe enough to reduce the hemoglobin level to 7.5 gm. or less. The level of 7.5 gm. was chosen because at this level of hemoglobin, with the usual associated reduction in blood volume, ischemic renal injury was imminent. We believe that the foregoing criteria of massive hemorrhage are applicable under almost any circumstances. Our cases of massive bleeding from duodenal or gastric ulcer met these criteria and, excluding all cases with unproved diagnoses of gastric or duodenal ulcer, seventy-three cases

occurred during the one year. The causative presence of gastric or duodenal ulcer was proved in each case by x-ray studies, surgery or autopsy.

Erythrocyte counts have not been included since it has been well established that such counts are subject to an error of plus to minus 20 per cent. Likewise, hematocrit determinations were not included since a high speed centrifuge is not available in many institutions. However, the results of this determination are accurate within 1 per cent [5].

BASIC MANAGEMENT

All patients were treated initially with aggressive nonoperative means. These consisted of vigorous ulcer therapy and rapid restoration of blood volume. The former included the liberal use of parasympatholytic drugs, sedation, Levin tube with suction if vomiting was present, alternate hourly feedings day and night, and oral antiacid drugs if nausea and vomiting were absent. Because re-bleeding is probably due to peptic digestion of the clot, it is of the greatest importance that the feedings be continued during the night. This is equally true of the substances used to reduce acidity and thereby to inhibit pepsin activity. The blood deficit was restored with whole blood, each transfusion consisting of 500 cc. of whole blood and 120 cc. of anticoagulant solution. The goal with transfusion therapy was to restore the hemoglobin to near normal levels rather than merely to alleviate the symptoms and signs of shock.

Roentgenographic examination was not performed routinely during the period of active bleeding. Our decisions were made primarily on the basis of the patient's history, physical examination, laboratory data, and the changes in these while the patient was under treatment. The problem of diagnosis is not considered in this report.

INDICATIONS FOR OPERATIVE TREATMENT

Two indications for surgery were used during the year that these patients were treated. The analysis to be presented was carried out mainly to determine the validity of these indications as a basis for surgical intervention in massive bleeding from peptic ulcer. These two indications were: *continued bleeding*, or *one episode of major rebleeding*. Continued bleeding necessitating surgery was considered to be present if the patient had signs and symptoms of shock despite the administration of 1,000 to 1,500 cc. of blood. With this indication it may be that the ulcer actually is bleeding intermittently, instead of continuously. Frequently under these conditions, active bleeding will not be present at the time of surgery. However, the clot usually can be easily dislodged. The second indication, one episode of severe re-bleeding, is more precise. Fundamentally, these indications are based on the large mass of evidence that, even with generous blood replacement, patients with repeated hemorrhages become progressively poorer risks [4,6,7,8]. Two gastric ulcer patients and three of the duodenal ulcer patients had emergency operations because of continued bleeding. Eight of the patients with gastric ulcer and seven of the patients with duodenal ulcer were operated upon because of severe re-bleeding. Major re-bleeding nearly always occurred within three to four days after hospitalization. This observation is similar to that made by Mathewson [2] and Welch [3] in similar groups of patients.

TABLE 1
SUMMARY OF MANAGEMENT OF CASES AND RESULTS

Data	Cases	Deaths
Duodenal ulcer:		
Operation	10	2 (20%)
No operation	49	6 (12.3%)
Gastric ulcer:		
Operation	10	2 (20%)
No operation	4	3 (75%)
Total	73	13 (17.8%)

REVIEW OF CASES

Table 1 shows a summary of the management of these cases. It is quite obvious that the mortality rate from bleeding gastric ulcers is much higher, and that emergency surgery is more often necessary, than is the case in bleeding duodenal ulcers. Mathewson and Welch suggest that this is due to the fact that gastric ulcers are more prone to erode into major arteries while duodenal ulcers more often cause bleeding from small vessels [2,3]. Another factor may be the relatively low acid values in gastric ulcer compared to those in duodenal

TABLE II

CORRELATION OF AGE, RACE AND SEX WITH MORTALITY

Data		Gastric Ulcer		Duodenal Ulcer		Total	
		Cases	Deaths	Cases	Deaths	Cases	Deaths
Age	20–49	2	1	17	0	19	1
	50–59	2	0	18	2	20	2
	60–88	10	4	24	6	34	10
Race	Caucasian*	12	4	45	8	57	12
	Negro	2	1	14	0	16	1
Sex	Males	8	3	48	8	56	11
	Females	6	2	11	0	17	2

* Includes Mexicans

ulcer. These lower acid values very possibly reduce the effects of medical treatment of the acid pepsin mechanism. In this series of cases, ten of fourteen patients with gastric ulcers were over sixty years of age and four of the ten patients died. By contrast, only twenty-four of the fifty-nine patients with duodenal ulcer were over sixty years of age and six of the twenty-four patients died. It is likely that the more advanced age of the patients with gastric ulcer is the major determining factor in the mortality difference between the two groups of patients. A more detailed study of the age distribution of all cases (Table II) reveals that almost three-fourths of the patients were over fifty years of age. Ten of the thirteen deaths occurred in the thirty-four patients over sixty; two deaths occurred in the twenty patients in their fifties; and only one death occurred in the group under fifty years of age. These mortality figures clearly indicate the grave danger associated with massive hemorrhage in the older age groups, as has been emphasized by Allen [9] and Welch [3,4]. However, this should not be interpreted as implying a high degree of safety in younger patients with massively bleeding peptic ulcer, because only nineteen of our seventy-three cases were under fifty years of age. There may be a high correlation between the age of the ulcer and the seriousness of the bleeding. Thus, a long standing, deeply eroded ulcer in a younger patient may present a very grave danger. Such ulcers probably increase in frequency in older age groups and further condition the increased seriousness of their bleeding episodes.

The great majority of the cases, and all but one of the deaths, occurred in Caucasians. During the period of this study, the average daily patient census of the Los Angeles County General Hospital included about 31 per cent Negroes and 20 per cent Mexicans; yet of these massive bleeders, only 22 per cent were Negroes and only 4 per cent were of Mexican descent. A study of Table II also confirms the great predilection of duodenal ulcer disease for males. However, the gastric ulcers were almost evenly divided among the sexes, and the two deaths among the female patients occurred in patients with gastric ulcer. Nevertheless, females and Negroes had a very low mortality rate.

A critical evaluation was made of each patient's history, physical examination, and laboratory data in an attempt to elicit some factors of prognostic value. While the facts are informative, little of statistical prognostic significance is apparent. (Table III). The mortality rate in those patients who had previously suffered pain or bleeding from an ulcer was not significantly different from that of those who had not had these symptoms. Furthermore, the presence of shock at the time of admission, the occurrence of hematemesis, or the previous demonstration of an ulcer by x-ray, did not correlate with an increased mortality rate. The occurrence of shock from re-bleeding after the initiation of hospital treatment, was of major and ominous prognostic significance. Of the thirty-two patients in whom this occurred, twelve died.

BLOOD REPLACEMENT

Of the 544 transfusions given to these patients, 168 were given to the fourteen patients

TABLE III
CORRELATION OF SIGNS AND SYMPTOMS WITH MORTALITY

Data	Gastric Ulcer		Duodenal Ulcer		Totals	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Previous ulcer pain	8	2	44	6	52	8
Previous ulcer hemorrhage	10	1	27	6	37	7
Acute hematemesis	11	4	36	4	47	8
Shock on admission	10	3	45	7	55	10
Shock after treatment	11	4	21	8	32	12
Ulcer on old x-rays	2	0	13	2	15	2

with gastric ulcers, an average of twelve transfusions per patient. One hundred twenty-eight transfusions, 12.8 per patient, were received by the ten patients with duodenal ulcer who had had emergency surgery. The forty-nine patients with duodenal ulcers who were not operated upon averaged slightly more than five transfusions each; only fourteen in this group received more than five. (Table IV.) In the entire group there was no patient who subsequently returned with hepatitis. Also, there was no recognized instance of hemolytic transfusion reaction, or of overtransfusion. Furthermore, there was no incident from which it could be concluded that restoration of normal blood pressure had been responsible for dislodgment of the clot from a vessel. Evidence against such an effect has been provided by Stewart's clinical study [1] and by the experimental study of Mayo [10].

TABLE IV
NUMBER OF TRANSFUSIONS

Data	Cases	Average No. Transfusions/ Patient	Total
Gastric ulcer	14	12	168
Duodenal ulcer (operated)	10	12.8	128
Duodenal ulcer (not operated)	49	5.1	248

IMPORTANCE OF BLOOD TYPE

In conjunction with this analysis of massive bleeding, the blood types were recorded in an additional group of patients with gastric or duodenal ulcer. Summaries are presented in Table V of 183 patients with duodenal ulcer and forty-nine with gastric ulcer. There was no difference in the blood type between the groups with or without massive hemorrhage. Here also, is shown the incidence of these blood types obtained from the Los Angeles Red Cross Blood Bank, and also from the blood bank of the Los Angeles County Hospital. There is evidence of a highly disproportionate percentage of patients with duodenal ulcer who are blood type O. The variations shown in our cases are in agreement with those reported from England by Koster [11] and Brown [12].

TABLE V
DISTRIBUTION OF BLOOD TYPES

	O	A	B	AB
Duodenal ulcer (Los Angeles County General Hospital 183 cases)	65.5	26.8	6.4	1.3
Gastric ulcer (Los Angeles County General Hospital 49 cases)	47.8	43.5	4.3	4.3
Los Angeles Red Cross donors	44.9	38.7	12.3	4.3
Los Angeles County General Hospital donors (10,199)	51	34	12	3

No analysis was made of the Rh factor. However, this factor, when negative in an ulcer patient with type B or AB blood, may involve major procurement problems if massive hemorrhage occurs. There are times in the city of Los Angeles when AB negative and B negative blood cannot be found in any quantity. Therefore, it is possible that a patient with chronic peptic ulcer, who is a B or AB Rh negative,

presents a relative indication for elective surgery on the basis of this difficulty. In addition the indication for emergency surgery logically will be much less rigid with an individual who is an AB Rh negative or B Rh negative. These considerations are of importance even in AB and B types who are Rh positive.

TABLE VI
MAJOR ASSOCIATED ILLNESSES
(46 Patients)

Chronic alcoholism	14
Cirrhosis	4
Hypertensive cardiovascular disease	13
Arteriosclerotic heart disease	7
Chronic renal disease	4
Cancer	3
Psychosis	2
Neurosyphilis	2
Diabetic coma	1

Although massive blood replacement was used in many of the patients in our group, no coagulopathies were noted except in one case which is to be mentioned later. This lack of tendency to induced bleeding may be related to the high rate of turnover and the resultant freshness of blood in the Los Angeles County Hospital Blood Bank. However, in private hospitals where blood is procured from a central facility, the blood supplied to patients is frequently more than ten days old. The physician ordering blood for his patient generally is unaware of this fact. If massive transfusion therapy becomes necessary, every effort should be made to assure the use of recently drawn blood. We have consistently found blood drawn two weeks previous to use to have a high serum potassium content and to be very thrombopenic.

ANALYSIS OF DEATHS

Forty-six of the seventy-three patients had at least one very serious complicating illness, (Table VI), heart disease and alcoholism being the most prevalent. In three patients (slightly over 4 per cent of the total cases) the primary cause of death was a complicating illness. It seems reasonable to postulate that in any similar group of patients, possibly 4 to 5 per cent would be so seriously ill from associated disease or moribundity, that they would die soon after admission or, at least, would not be acceptable candidates for surgical intervention.

In evaluating the deaths in our cases, a decision has been made in every instance as to the inevitability of death. These decisions were made after exhaustive review and discussion of each record. If it was decided that a death might have been prevented by surgical intervention. This decision was based on whether or not there existed one of the indications previously stated (continued bleeding or one severe re-bleeding episode). If the death was considered to be due to some other error, the analysis indicates this, and the death is considered preventable. Only by such an analysis can the adequacy of the indications be investigated and other errors be revealed.

DUODENAL ULCER

Operation. Ten patients with duodenal ulcer had emergency operations, resulting in two deaths. Of these, one death might have been avoided and one was unavoidable. The avoidable death occurred in a sixty-four year old man who was allowed to re-bleed twice, necessitating twelve transfusions before surgery. This was in violation of the previously stated policy. Postoperatively, signs and symptoms of peritonitis developed, and he died despite intensive treatment. An autopsy was not performed. This death is considered to be avoidable for two reasons: (1) The patient should have been operated upon earlier and (2) there was apparently some technical failure productive of the postoperative abdominal signs and symptoms. The unavoidable death occurred in a seventy-seven year old man who continued to bleed after surgical intervention. He had very serious heart disease, diagnosed as severe aortic stenosis, and regurgitation with congestive failure. A pyloroplasty with suture ligation of the bleeding vessels was performed. The patient died of cardiac failure three days postoperatively, and at necropsy there was no evidence of gastrointestinal bleeding.

Varied technical procedures were utilized in the ten duodenal ulcer cases in which operations were performed. Four patients had subtotal gastric resections with gastrojejunal anastomoses; two patients had vagotomy, pyloroplasty and sleeve gastric resections; two had vagotomy and pyloroplasty, one patient had a subtotal gastric resection with a gastroduodenal anastomosis; and one patient had a pyloroplasty only. Suture ligation of the bleeding vessels was done in all cases.

The morbidity in this group of cases was small, with no evidence of anastomotic leaks or stomal malfunction. One patient had a severe wound infection followed by an incisional hernia.

No Surgery. There were forty-nine patients with duodenal ulcer who were not operated upon. In this group there were six deaths, two avoidable and four unavoidable. The first of the two avoidable deaths occurred in a fifty year old man with long standing hypertensive cardiovascular disease. He had a hemiplegia as the result of an old cerebrovascular accident but he was able to care for himself. He had been hospitalized because of lobar pneumonia from which he had recovered satisfactorily. Despite massive hemorrhage with continued bleeding, he was treated medically. The surgeons were never called in to see him. He died five days after the onset of hemorrhage and, at autopsy, there was an opening in a duodenal vessel from which he had exsanquinated. The second death in this group was of a seventy-six year old man who had mild arteriosclerotic heart disease and re-bled under therapy, but who was not operated upon. He succumbed to the second bleeding episode. Using our indications, both of these patients should have had emergency surgery and are therefore classified as potentially preventable deaths.

In the unavoidable death group was a seventy-eight year old man who refused to undergo an operation. The second unavoidable death occurred in an eighty-two year old man who entered the hospital moribund. The third fatality was a fifty year old chronic alcoholic, with severe cirrhosis and a bleeding ulcer, who was also moribund upon admission. The latter two patients died within an hour of arrival at the hospital. The fourth patient was suffering from severe congestive heart failure. He re-bled and died very quickly.

GASTRIC ULCER

Surgery. Of the fourteen patients with gastric ulcers, ten had emergency operations resulting in two unavoidable deaths, and four patients were not operated upon, with one avoidable death and two unavoidable deaths. The first unavoidable death postoperatively occurred in an eighty-eight year old woman with hypertensive cardiovascular disease and congestive failure, who was operated upon after she re-bled. Postoperatively she continued to be in heart failure and finally died on the fifth postoperative day. At no time was there any evidence of recurrent bleeding. The second unavoidable death was in a sixty-one year old man who had two separate bleeding episodes and two operations. After the first massive hemorrhage he re-bled and had a wedge excision of a high-lying lesser curvature gastric ulcer about 2 cm. in diameter. His condition was satisfactory for two months, after which he returned with another massive bleeding episode. This time he continued to bleed after admission and underwent an emergency laparotomy, at which time he was found to have a juxtaesophageal, posterior wall gastric ulcer, 6 cm. in diameter, which had eroded 2 cm. into the pancreas. During the operation signs of a coagulopathy developed which were determined to be due to hypofibrinoginemia. The patient received twenty-nine transfusions, intravenous fibrinogen and underwent a subtotal gastric resection, despite which he died on the third postoperative day in irreversible shock. Although his death was considered to be unavoidable at the time of his second admission, the death might well have been prevented by a subtotal gastrectomy at the first operation.

The operative procedures used on these ten patients were wedge excision in three cases and subtotal gastrectomy with gastrojejunostomy in one case. The remainder were treated by subtotal gastrectomy with gastroduodenostomy. Except for three wound infections, the morbidity among the surviving patients was not significant.

No Surgery. The death of one of the patients with gastric ulcer who was not operated upon should have been avoided. He was a forty-eight year old man who had a massive hemorrhage after being admitted and re-bled twice. Each time he recovered very quickly with the treatment that was utilized and, despite our established policy, he was not operated upon. He exsanguinated in his sleep on the fifth night after admission; necropsy showed a gastric ulcer with erosion into a vessel with the gastrointestinal tract full of blood.

The last two patients whose deaths were unavoidable (using our operative indications) were a sixty-three year old woman and a seventy year old man. Both had severe hypertensive cardiovascular disease and were under cardiac therapy when they had their massive hemorrhages. Both patients made a rapid

and satisfactory response to non-operative treatment initially and were considered to be out of danger. About twelve hours after admission in each case, torrential hematemesis suddenly developed and the patient exsanguinated within twenty or thirty minutes.

COMMENTS

Utilizing a policy of treatment for massive gastroduodenal hemorrhage consisting of vigorous ulcer therapy and restoration of blood volume, with emergency surgery being reserved for those patients who continued to bleed or who re-bled, we had a mortality rate of 17.8 per cent. If the four deaths we considered avoidable could have been prevented, we would have had a mortality rate of 12.3 per cent (nine unavoidable deaths in seventy-three cases). This would then indicate a maximal possible salvage of 87.7 per cent of cases, under the conditions presented and using the operative indications that we described.

What would have been the theoretic optimal mortality rate if, during this period, a policy had been followed of immediate emergency surgery for all patients with massive bleeding who were not rendered inoperable because of severe systemic disease or moribundity? In this series, nearly 5 per cent of the patients were inoperable for these latter reasons, and it seems valid to assume that this would be true in any similar series. If we could postulate an operative mortality rate of 5 per cent, which seems lower than it probably would be in a group such as ours, the total mortality rate would be 10 per cent. Therefore we believe that the difference between the two theoretic mortality rates (12.3 per cent versus 10 per cent) and our actual mortality rate of 17.8 per cent is not significant enough to justify a change in our policy. In the hands of a select group of very skillful surgeons, under ideal hospital conditions, the more radical policy might be successfully employed.

In contemplating the operative procedure to be used in these cases, it seems to us that the surgeon must assume that his primary objective is to stop the hemorrhage in order to save the patient's life. Therefore, the most expeditious technical procedure possible should be utilized to control hemorrhage and to prevent recurrence. This means that frequently, a procedure less radical than a 75 or 80 per cent subtotal gastric resection is justified.

CONCLUSIONS

We have defined massive hemorrhage from peptic ulcer as acute hemorrhage accompanied by hypovolemic shock with a systolic blood pressure of 90 mm. Hg or less and/or productive of a fall of hemoglobin to 7.5 gm. per 100 cc. or less. Generally usable criteria should be developed for all who attempt to evaluate results in the treatment of massive hemorrhage from duodenal or gastric ulcer. It is desirable that medical and surgical cases be reported, showing the total hospital mortality. On the basis of the data presented, it is our intention to maintain our present indications of continued bleeding or one severe re-bleeding for emergency surgical intervention, with emphasis on severe re-bleeding as an almost absolute indication for immediate surgery. The successful treatment of patients with massive hemorrhage from duodenal or gastric ulcer requires large volumes of blood (2,500 to 6,000 cc.). Since ten of the thirteen deaths in this series were in patients over sixty years of age, it is suggested that without exception, everyone who has a known active peptic ulcer, and especially if it be gastric, should have appropriate surgical treatment before his or her sixtieth birthday. Furthermore, the duration of an ulcer should become a factor in determining the need for elective surgery. Finally, ulcer patients with AB or B type blood, especially if Rh negative, are candidates for earlier elective and emergency surgery.

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DISCUSSION

HERBERT J. MOVIUS (Long Beach, Calif.): I agree with the authors on several important points. Certainly the necessity for a set of rigid criteria, defining massive hemorrhage, is mandatory if anything of value is to come out of the great mass of hospital charts being perused by surgeons throughout the country. The authors' definition of massive hemorrhage is excellent and is particularly valuable in that no out-of-the-ordinary clinical laboratory test is needed. Few if any patients would be erroneously included as having "massive hemorrhage" if these criteria were adhered to. It would be a real step forward if some such simple set of criteria could be nationally adopted, at least for future clinical analyses.

I certainly agree that the blood volume should be restored to approximately normal in the patient having recently manifested severe hemorrhage and that mere correction of hypovolemic signs and symptoms should not be the stopping point for blood replacement. Re-bleeding, as was emphasized by the authors, should be a positive indication for surgical intervention. This is one condition which certainly should jar the physician into action and should end any procrastination on the part of the surgeon concerning when to operate. It is unfortunate, however, that internists and some surgeons allow re-bleeding to continue on medical therapy, as evidenced in this manuscript by the report of thirty-two patients who manifested re-bleeding and only twenty of whom were operated upon.

At the Long Beach Veterans Administration Hospital we have vigorously taught the residents that the prime concern of the surgeon operating upon a patient for massive hemorrhage is to stop that bleeding. Certainly there can be no brief with the authors when they state that many times these patients may not be candidates for more extensive definitive ulcer procedures. At the Long Beach Veterans Administration Hospital we have frequently used the procedure of transfixion suture of the bleeding ulcer and supplemented this with vagotomy and pyloroplasty. In twenty-six consecutive cases we have had but one death. This is too small a number from which to draw conclusions, but if the phrase may be used in a non-election year, "it shows a trend."

Finally, I was pleased to see the authors' suggestion that patients with chronic gastroduodenal ulcer be submitted to elective surgical management prior to their attaining the age of sixty. This, I believe, is a true forward step in the over-all management of patients with peptic ulcer and I wonder if we should not adopt a far more liberal set of indications for surgical intervention in the management of all patients with peptic ulcer than the now "well accepted criteria." It is, of course, difficult to state the mortality rate of patients with peptic ulcer for the lifetime of the patient on a medical regimen, but it certainly must be close to 5 per cent. In addition, the regimen is confining, it does not allow many of the patients to freely participate in what is considered to be a well rounded social scheme, the regimen is highly atherogenic (although this might be splitting hairs) and, finally, it is expensive and time consuming when considered over a period of years.

If a definitive operation to combat the ulcer diathesis can be accomplished with a mortality rate of one-half of 1 per cent, and this has been the mortality figure for vagotomy and pyloroplasty in over 1,000 cases at the Long Beach Veterans Hospital, than why should not such an operation be offered to the patient with peptic ulcer before he bleeds, perforates, obstructs or becomes totally refractory to vigorous medical management.

I disagree with the authors on a few trivial points. We have found the use of an upper gastrointestinal series, with or without the Hampton technic, to be very valuable in all massive upper gastrointestinal hemorrhage, except those patients in shock. The roentgenogram may reveal a gastric ulcer or a giant duodenal ulcer, which in either case should call for surgical intervention without delay. I also question the wisdom of attempting to classify deaths as avoidable. This could open avenues for conflicting statistical analyses and may tend to negate other well accepted rigid criteria. Finally, the statement "surgery is indicated as bleeding continues" is ambiguous. I personally have no formula or method for determining when surgery is indicated and how to determine when bleeding is, in fact, continuing. We have all had experiences that show that a fairly high percentage of patients who are operated upon for continued bleeding, are in truth, not actively bleeding at the time the ulcer is viewed. Therefore, I would appreciate, but have no suggestion myself, an accurate end point in the clinical titration of the patient who is admitted

with active bleeding. When do we make the decision to operate?

EDWARD B. SPEIR (Seattle, Wash.): I thought it might be of some interest to let you know that one of the residents and I recently went over the statistics at the Swedish Hospital for a two-year period. We grouped the patients who were coded and diagnosed on entering the hospital as having gastrointestinal hemorrhage.

Three hundred three patients were seen during the two-year period. Seventy-eight per cent of the patients were treated medically, with a mortality rate of 11.5 per cent.

Eighteen per cent were treated surgically, which meant sixty-eight patients, with a mortality of 11.7 per cent.

The other interesting fact was that of the over-all 303 patients, twelve continued to bleed and were operated upon, as far as we could determine, while still bleeding. Of these twelve patients, three died, giving for that particular group a 25 per cent mortality rate. We tell the medical people, of course, that there is a 75 per cent salvage rate, but it only points out the seriousness of the disease.

The other thing which interested me in Drs. Berne and Snyder's report was that our use of blood paralleled theirs quite closely. It varied in the surgical patients from three to thirty-one units, with an average of eight.

The Medical Service supplied their patients, in the range from one to thirty-three units, with an average of 3.2 units of blood.

EDWARD N. SNYDER, JR. (closing): In answer to the remark by Dr. Movius about continued bleeding, to a certain extent we left the term ambiguous because continued bleeding means various things to various people. I do not see how anyone can say for certain that a given patient has a continued minor hemorrhage.

Of the total group of patients who were operated upon for what was considered to be continued or recurrent bleeding, that is all twenty patients, twelve were found to have arterial hemorrhage at the time of surgery.

Of the people who were operated upon for what was thought to be just continued bleeding only, three had arterial hemorrhage at the time of surgery.

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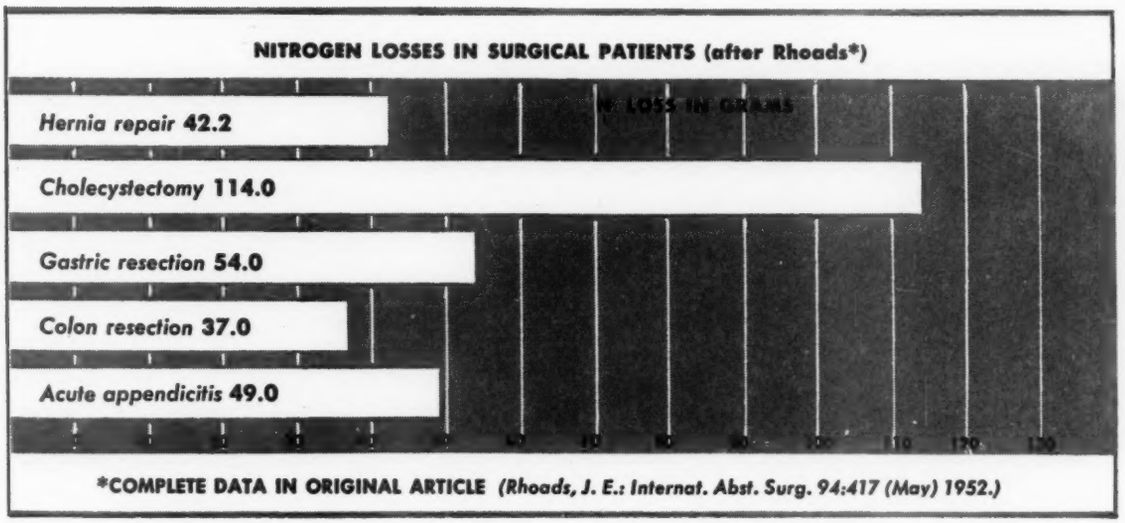
NITROGEN LOSSES IN SURGICAL PATIENTS (after Rhoads*)
N LOSS IN GRAMS
Hernia repair 42.2
Cholecystectomy 114.0
Gastric resection 54.0
Colon resection 37.0
Acute appendicitis 49.0
*COMPLETE DATA IN ORIGINAL ARTICLE (Rhoads, J. E.: Internat. Abst. Surg. 94:417 (May) 1952.)

Patients about to undergo extensive surgery[1] frequently have negative nitrogen balance and protein deficiency. And after any severe trauma, including extensive surgery, the rate of protein breakdown is increased.

It is also well recognized that patients with a strongly negative nitrogen balance are much more prone to suffer delayed wound healing[2], secondary infections[3], shock[2] and delayed convalescence[4].

The need for an effective protein anabolic agent is stated by Moore and Ball[5]—"there is one unbreakable rule of surgical convalescence: to complete his recovery, regain strength and return to work the patient *must* come into positive nitrogen balance."

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1. Flocks, R. H.: J.A.M.A. *163*:709 (Mar. 2) 1957.
2. Flocks, R. H.; Marberger, H.; Begley, B. J., and Prendergast, L. J.: J. Urol. *74*:549, 1955.

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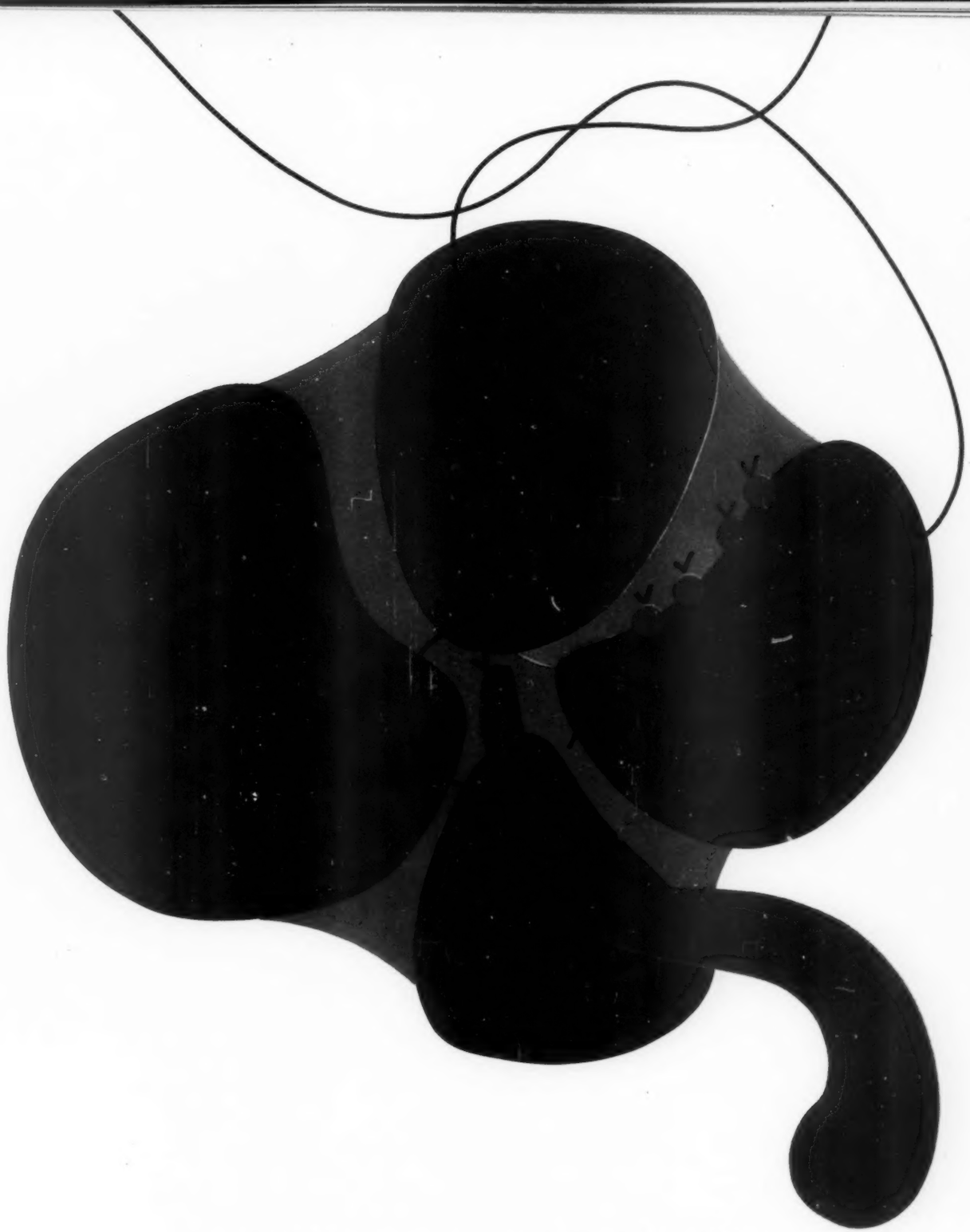

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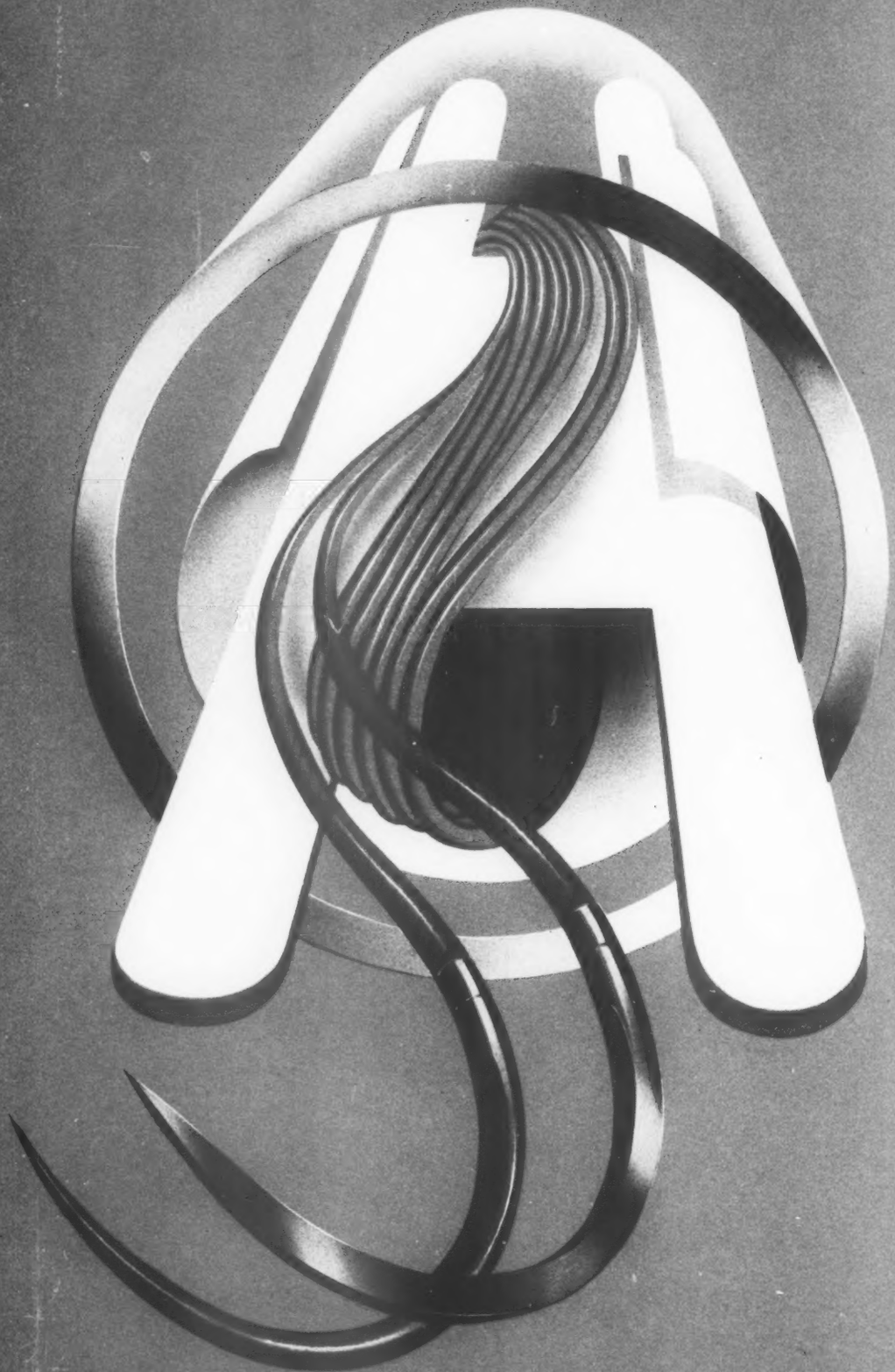

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*Elman, R.: *Protein Needs in Surgical Patients, J. Am. Dietetic Assn. 32;524 (June) 1956.*

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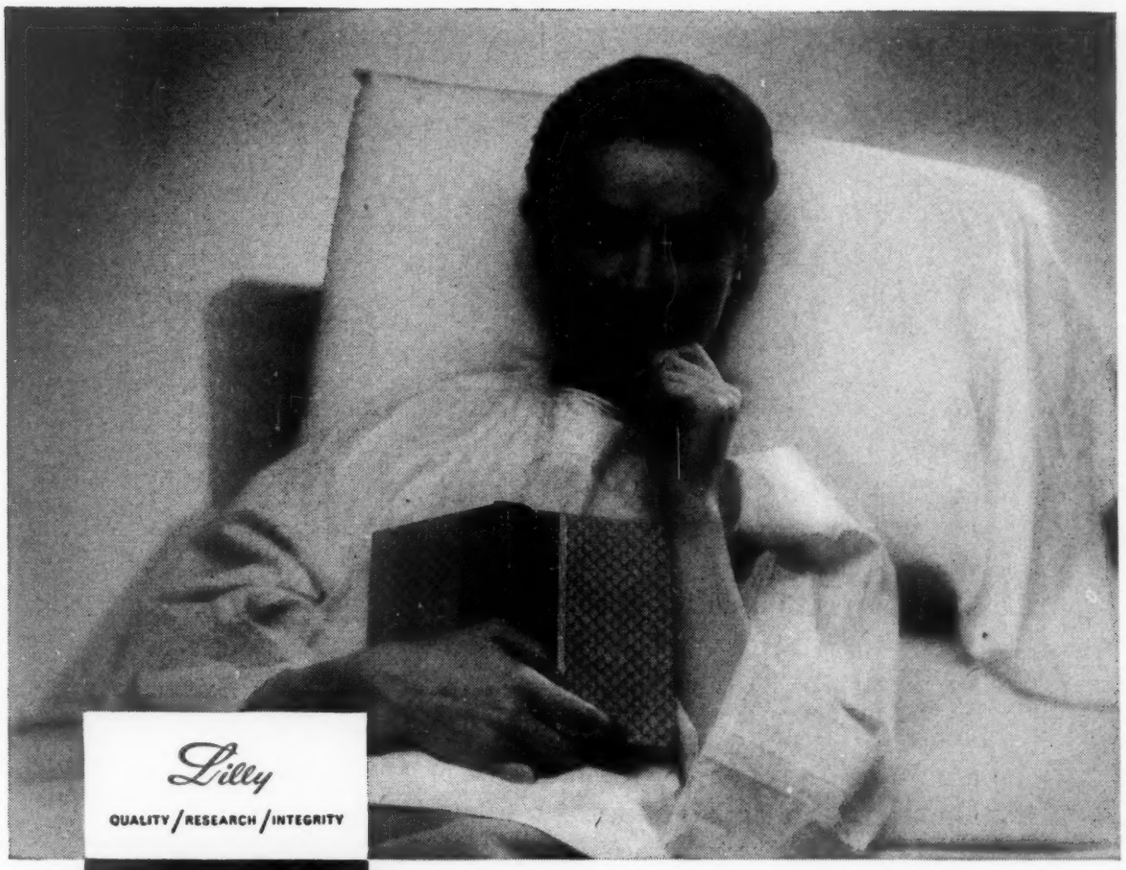

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1. Proudfit, P. T., and Robinson, C. H.: Nutrition and Diet Therapy, ed. 11, New York, The Macmillan Company, 1955, pp. 314-320.
2. Harper, A. E.: Amino Acid Imbalance, Toxicities and Antagonisms, Nutrition Rev. *14*:225 (Aug.) 1956.
3. Amino Acid Requirements of Adult Man, Nutrition Rev. *14*:232 (Aug.) 1956.
4. Amino Acid Imbalance and Supplementation, Editorial, J.A.M.A. *161*:884 (June 30) 1956. Council on Foods and Nutrition, American Medical Association: Importance of Amino Acid Balance in Nutrition, J.A.M.A. *158*:655 (June 25) 1955.

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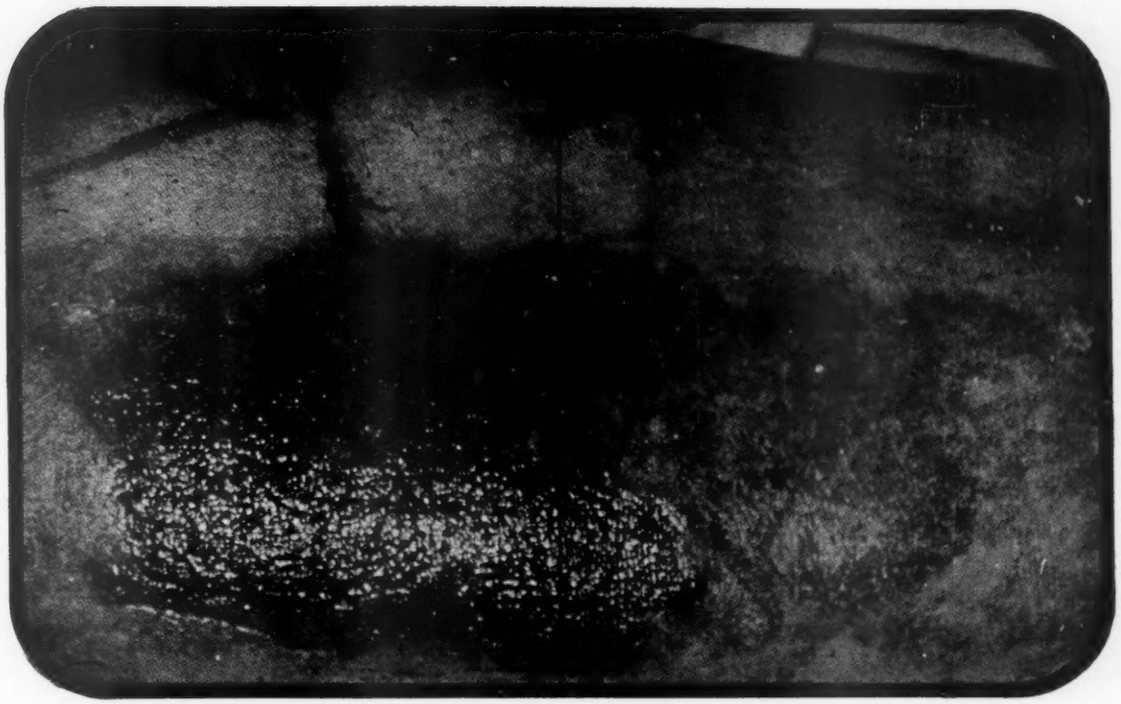

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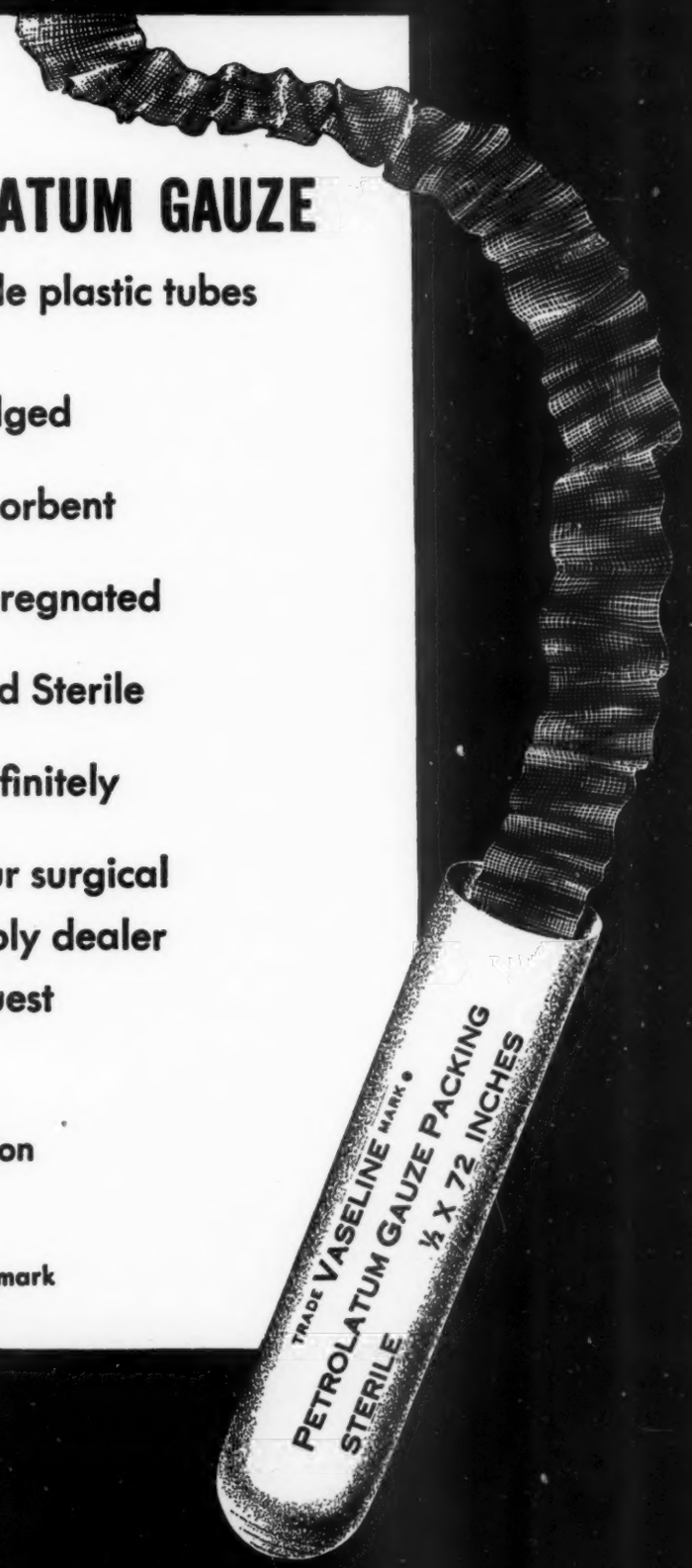

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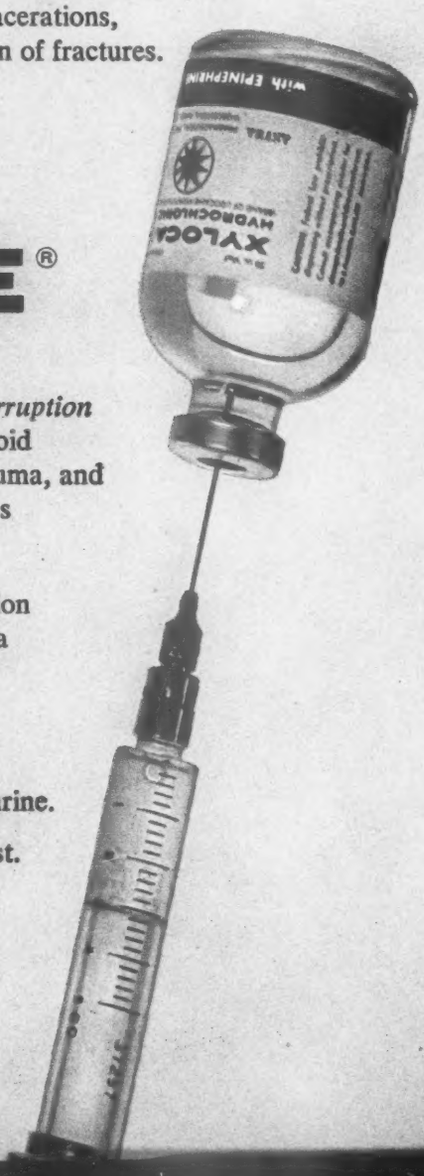

*Southworth, J. L., and Dabbs, C. H.: Xylocaine: a superior agent for conduction anesthesia, Anesth. & Analg. *32*:159 (May-June) 1953.

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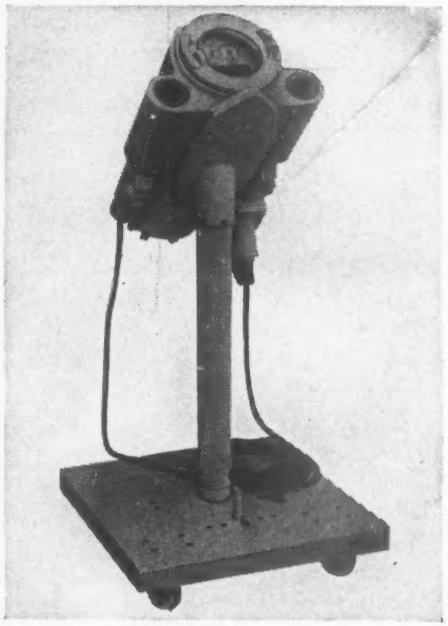

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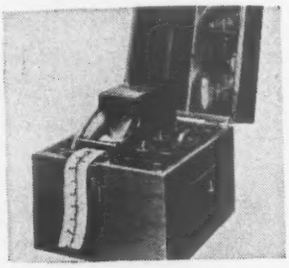

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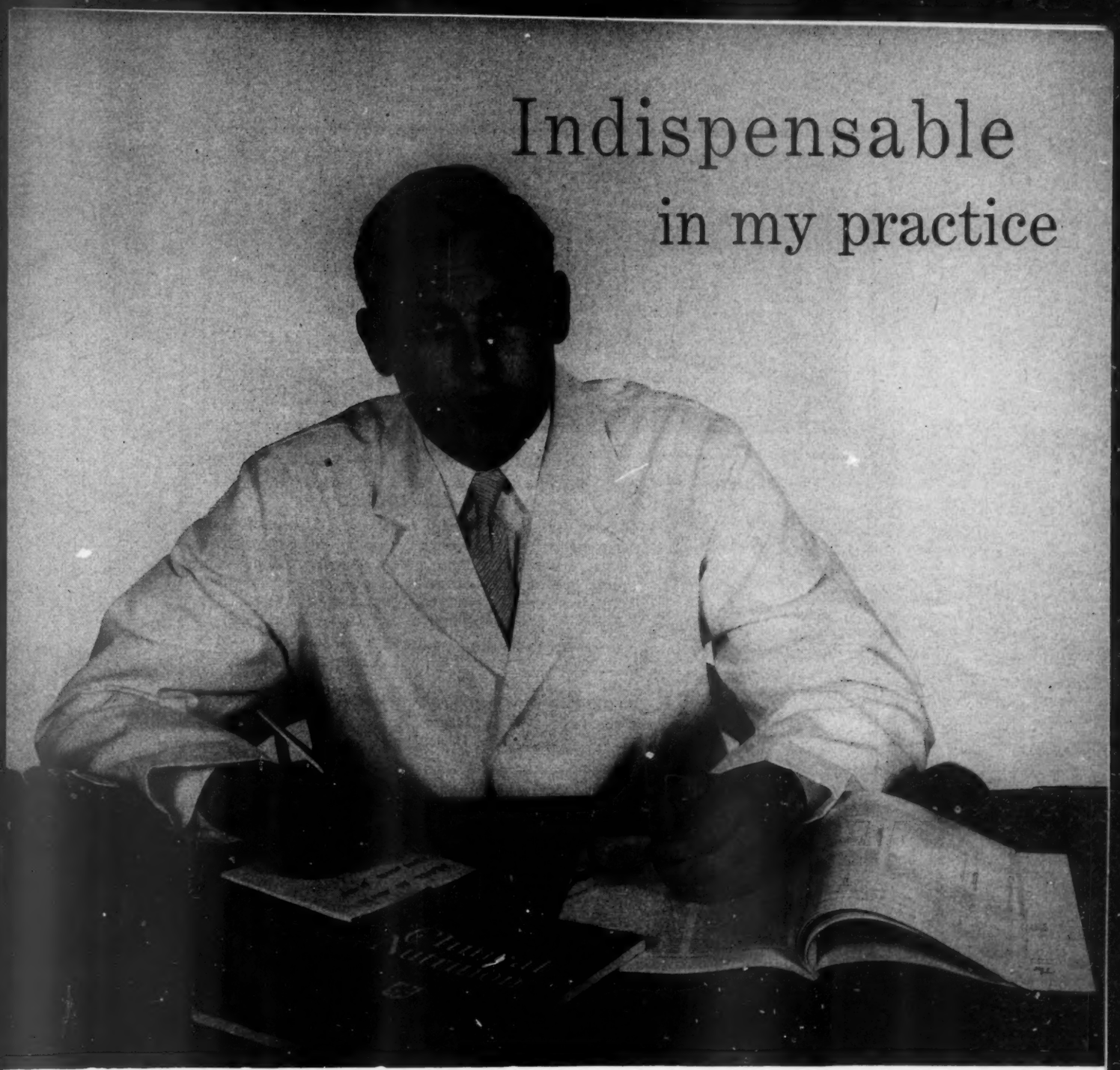

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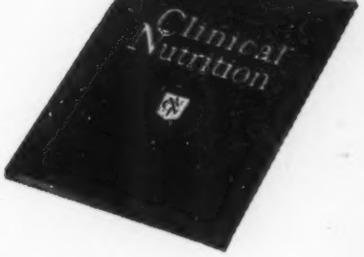

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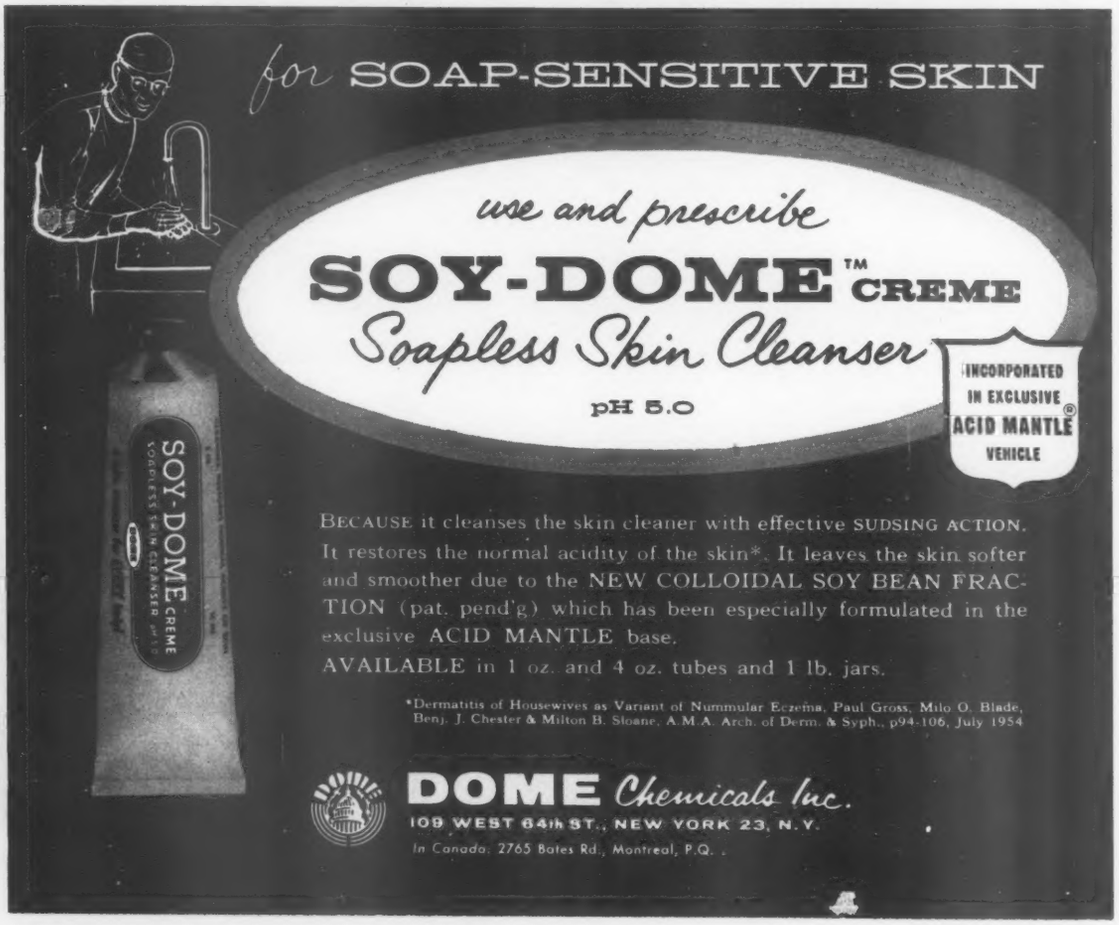
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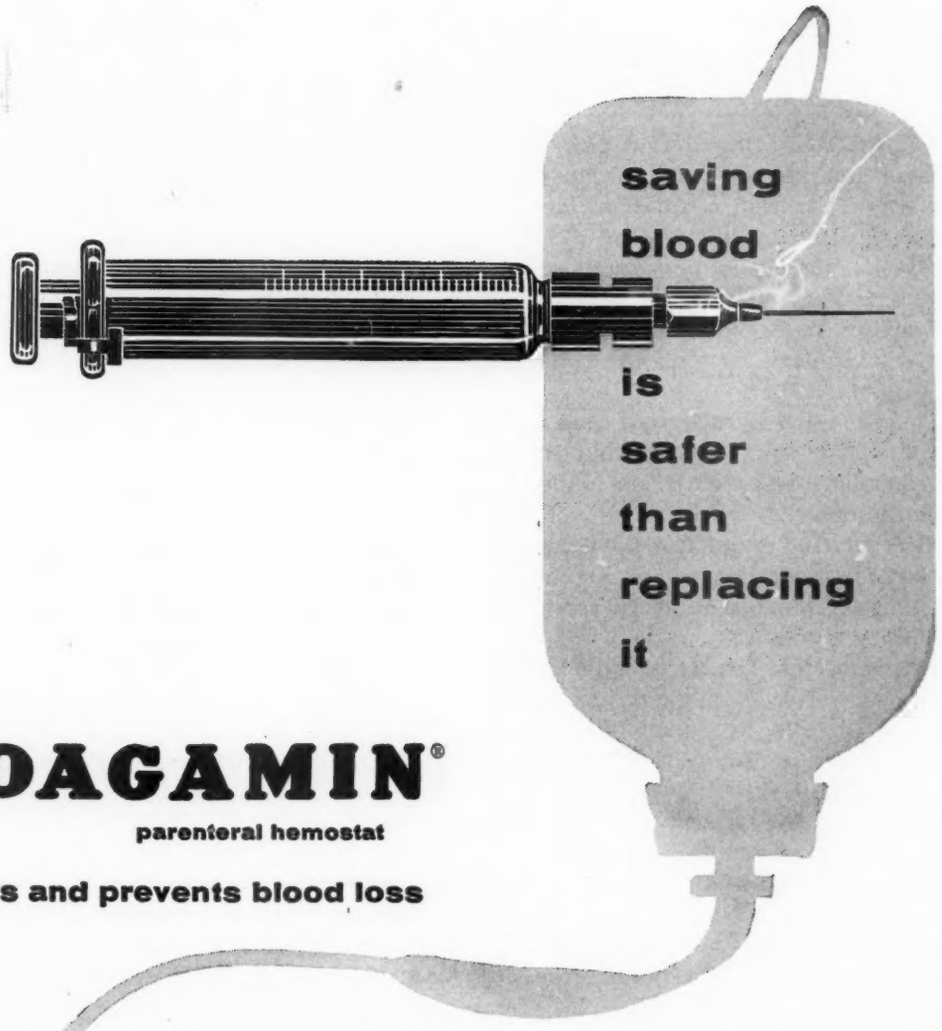

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1. Joseph, M.: Control of Hemorrhage—or Transfusion, Am. J. Surg. *87*:905, 1954. 2. Crisp, W. E.: Editorial; One Pint of Blood, Obst. & Gynec. *7*:216, 1956.

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NOTES AND QUOTES

SUCCESSFUL transplanting of four parathyroid glands from one person to another at the Veterans Administration hospital in Houston, Texas, was reported by the hospital recently.

The rare surgery was performed by Dr. George L. Jordan, Jr., Chief of Surgical Service at the Houston V. A. hospital and Assistant Professor of Surgery at Baylor University Medical School.

He transferred the parathyroid glands of a stillborn baby to a thirty-six year old veteran, and the transplants apparently are functioning in a normal manner.

The entire thyroid gland to which the parathyroids were attached also was transplanted from the baby to the veteran at the same time, but it did not survive.

If the transplanted parathyroid glands continue to behave normally, most of the veteran's troubles may be over, because administration of artificial thyroid hormones is relatively simple, Dr. Jordan said.

According to medical literature, total transplantation of the thyroid and parathyroid glands has been attempted only three times previously, all at the Albert Einstein Medical Center in Philadelphia. The first was in 1952. Only two have been reported as being successful.

A remarkable change in the types of treatment for mental patients since tranquilizing drugs were started was reported by Veterans Administration recently.

Dr. J. F. Casey, V. A. Director of psychiatry and neurology in Washington, D. C., said the changes include: (1) *Electric shock treatment down from 4,527 patients in fiscal 1955 to approximately 1,000 patients for the first six months of fiscal 1957.* (2) *Insulin coma treatment cut from 1,486 patients in fiscal 1956 to only 383 patients for the first six months of fiscal 1957.* (3) *Neutral packs and tubs sharply reduced from 15,655 patients in fiscal 1955 to 1,471 patients for the first six months of fiscal 1957.* (4) *Privileged patients on tranquilizers increased from 2,500 in fiscal 1955 to 8,500 in the first half of fiscal 1957.* (5) *Trial visits away from the hospitals increased from 9,985 patients in fiscal 1955 to 12,351 in fiscal 1956.*

Dr. Casey said the tranquilizers also have caused a beneficial chain reaction in reawakening the interest of patients in individual and group psychotherapy, with the result that this form of treatment is on the increase.

"Perhaps one of the most dramatic changes has occurred in the number of patients who can be granted greater hospital freedom as a result of the drugs," Dr. Casey said. "More and more of them are being transferred from closed to open wards, which is a therapeutic advancement of considerable significance in the treatment of mental patients."

Dr. Casey said about half the psychiatric patients hospitalized by the V. A. are being treated with drugs. Patients receiving tranquilizers have increased from 17,696 in fiscal 1955 to more than 36,000 in the first half of fiscal 1957.

SWEDISH physicians have uncovered three new hereditary neuromuscular diseases, Dr. Gunnar Wohlfart, Chairman of the Department of Neurology at the University of Lund Medical School, Sweden, said at a University of Wisconsin Medical School lecture recently. The new diseases are proximal spinal muscular atrophy, distal myopathy and a new form of familial periodic paralysis.

Dr. Wohlfart said proximal spinal muscular atrophy is caused by nerve lesions with an effect much like muscular dystrophy. In the past, cases which were diagnosed as muscular dystrophy may actually have been this new disease, he said.

Distal myopathy was described by the neurologist as a disease which causes wasting of muscles in the hands and lower legs of persons over forty years of age. It was first discovered by Dr. L. Welander of Gothenburg, Sweden.

The classic form of familial periodic paralysis is known to be caused by decreased amounts of potassium in the blood, Dr. Wohlfart said. A new form of the disease, discovered by Dr. I. Gamsthorp of Lund, Sweden, is caused by increased amounts of potassium.

Dr. Gamsthorp found 138 cases of the new form in southern Sweden, but further investigation revealed that all cases were found among two families, beginning with a man who was born in 1599.

Dr. Wohlfart also reported on his studies of polio in mice and human beings. His experiments show that nerve fibers surrounding a

CONTINUED ON PAGE 50

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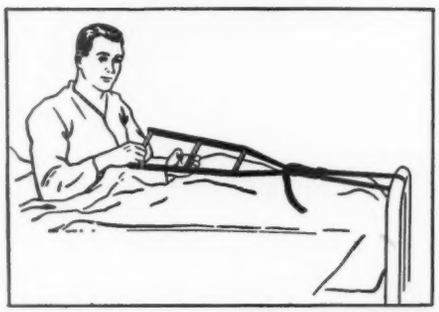

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destroyed nerve fiber branch out and revitalize the dead muscle area.

In mice, a muscle may regain up to 90 per cent of its strength within two months, even if one-half of its nerve fibers have been destroyed.

Recovery of muscles after an attack of polio had been previously linked exclusively to recovery of motor cells in the spinal column.

Dr. Wohlfart reported that, in human patients, an excess number of nerve branches were found in the muscles, indicating that surrounding nerve fibers had branched, as in mice. However, these fibers are often incapable of functioning normally and no way has yet been found to restore their capacity to transmit impulses.

As a means of meeting individual dosage requirements, Wyeth Laboratories now is marketing the tranquilizing drug equanil® in 200 mg. tablets as well as in the original 400 mg. size.

In the new reduced-strength tablets, the drug has proved a safe and orally effective agent for the relief of anxiety, tension, insomnia and muscle spasm. It relaxes both mind and body, does not affect autonomic functions, and has notably few side effects.

ASPIRIN in large amounts was a beneficial substitute during the withdrawal of adrenal steroids in treating five cases of pseudorheumatism, Drs. Jerome Rotstein and Robert A. Good state in the *Archives of Internal Medicine* (99: 545, 1957).

They describe the syndrome as a distinct clinical condition stemming from prolonged steroid treatment or rapid withdrawal of steroids following a period of therapy. The symptoms, which "mimic" rheumatoid arthritis, are often mistaken for increased arthritic activity.

Studies on five patients at the University of Minnesota Hospital disclosed that the two conditions can be differentiated by the site of the symptoms. In rheumatoid arthritis the predominant symptoms are synovitis and inflammation of the articular and periarticular tissues. The authors say the muscles and bones are the site of most pain in pseudorheumatism, and are disproportionately tender.

Gradual but persistent discontinuation of steroids was the treatment in all five cases. Much of the discomfort of withdrawal was relieved by large doses of aspirin, which often was the principal medication in succeeding months along with small doses of corticotropin to stimulate endogenous production of corticoids while the steroid was being withdrawn.

One of every fourteen boys and one of every seventeen girls under the age of twenty are hospitalized in the course of a year, the Metropolitan Life Insurance Company's statisticians report on the basis of the experience among dependent children of the company's personnel. The study concerns claims under group insurance on which the first payment was made during 1954 and 1955, and which involved at least one night of hospital stay.

Frequency of hospitalization was at the annual rate of sixty-nine per 1,000 for males and fifty-nine per 1,000 for females. Under the age of five, the rate for males was about 30 per cent higher than for females, whereas at ages fifteen to nineteen the rate for males was only slightly higher than the female rate.

The average length of hospital stay was five and a half days. Youngsters five through nine years of age had the shortest stay, averaging not much over three days. This is accounted for largely by the fact that tonsillectomies, which constituted the bulk of the cases at these ages, averaged only slightly more than one day in the hospital.

The fifteen through nineteen age group had the longest average stay, about 9 days.

Two-thirds of the hospitalized patients underwent surgery. Duration of the hospital stay was much shorter for the surgical patients than for the non-surgical, averaging less than half as long.

DRUG manufacturers cannot trust a preservative in a new product just because it was effective in another drug, an Abbott Laboratories microbiologist says in a paper on "Preservatives in Pharmaceutical Products."

"An agent may be effective in one product and fail in another," says Dr. Walton E. Grundy, Section Leader in Abbott's Department of Microbiology. "Unexpected incompatibilities occur often enough to make it difficult to predict its action."

Dr. Grundy presents data to show that benzyl alcohol is an excellent preservative in

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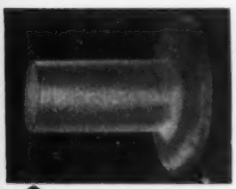

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some products. However, he points out, "These data do not imply that benzyl alcohol is a better preservative, for it will fail in certain products."

Preservatives, adequately tested in each new product, are essential both in sterile products for parenteral or topical use and in products which are not sterilized in the manufacturing process and which are mostly for oral administration.

In sterile products which are used repeatedly from the same container and may become contaminated, the preservative must prevent the growth of the contaminant. A preservative for oral medicinals is needed to maintain the stability and potency of the product by preventing growth of microorganisms which cause spoilage.

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Atraugrip jaws are a feature of many Pilling instruments which can be seen not only at the home office in Philadelphia but also at the new Pilling showroom at 4 West 56th Street, New York 19, New York.

A NEW instrument called the Freeze-Dry, made by the American Instrument Company of Silver Spring, Maryland, can be used to preserve living cells indefinitely without a need for refrigeration.

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Scientists at the United States Department of Agriculture use the Freeze-Dry to prepare simultaneously 100 samples of Brucella abortus culture for shipment. This culture is used for the inoculation of cattle against premature dropping of calves.

Scientists at the University of Maryland use the Freeze-Dry to preserve common cold virus for later study.

Leading tobacco companies are also using this instrument in research to isolate and eliminate irritants which may be found in tobacco.

Samples of the material one wishes to preserve or ship are quickly frozen in the integral freezing compartment of the instrument. They are then attached to the fixtures on the stainless steel drum and dried by sublimation (the boiling off and withdrawal of water from the material without the material first having been turned into liquid). The containers are flame-sealed to imprison the sample in a vacuum. They are then withdrawn without disturbing the drying of other samples. Normally the material is restored to its original state merely by adding water.

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Intermittent positive-pressure breathing and aerosol therapy using the bronchodilator isuprel® is warranted in all children with severe intractable asthma, according to Dr. Roy F. Goddard and Elizabeth H. Roorbach of the Lovelace Clinic.

Writing in the Journal of the American Medical Association *(163: 1125, 1957), they say the procedure was responsible for a "significant reversal of changes due to pulmonary impairment." They reported on a group of 100 children with asthma of varying severity who were treated over a four-year period.*

Ninety-eight per cent of the children, ranging in age from one to seventeen years, responded to intermittent positive-pressure breathing-aerosol therapy. Forty of the series had previously failed to respond to usual methods of therapy. The IPPB-aerosol method, they conclude, proved an "adjunctive, curative and prophylactic type of treatment."

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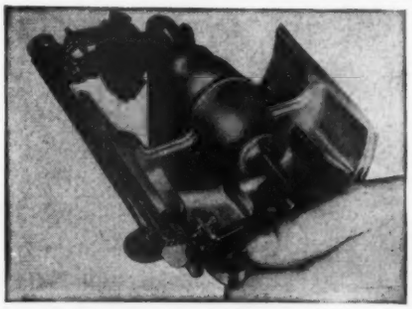

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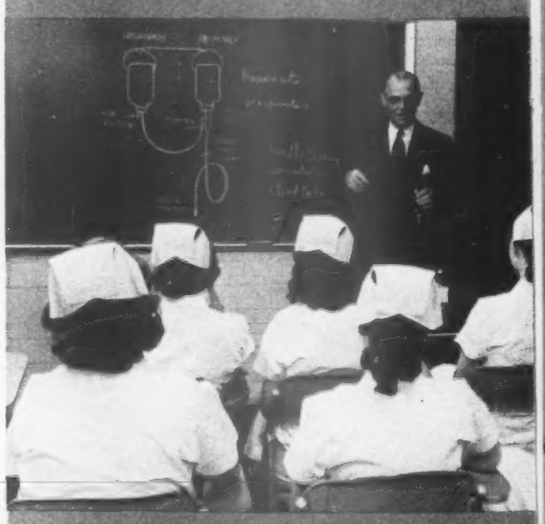

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